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EFFECT OF THE VOLATILE BASE IN FLUID INTESTINAL CONTENTS ON DOGS WITH LOW INTESTINAL OBSTRUCTION

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IN THE literature theories as to the cause of the symptoms and death in cases of mechanical obstruction are numerous. Some writers have accepted the dictum that a toxic substance was present in the intestine. As a result there have been thorough investigations of the fluid contents of the bowel to ascertain the existence of such toxins.

In a previous study one of the authors (J. S. H.)[5] showed that the gases occurring in low ileal obstruction could not cause symptoms of toxicity, provided their percentage was not greater than that found in simple obstruction.

The appearance of the volatile basic group of gases was entirely unexpected, since they had not been previously reported. Although this group of gases was harmless, added information consisting of an investigation of the volatile bases of the fluid contents, since they were in all probability the direct precursors of the gases, appeared to be important.

EXPERIMENTAL MATERIAL AND METHODS

The fluid contents of the obstructed intestines were collected immediately following death of the animal for analysis.

The total amount of volatile basic substance was determined by using a known amount of fresh contents from obstructed bowels and diluting with 200 c.c. of distilled water. To this was added 2 gm. of magnesium oxide to make the solution alkaline. The distillate from this mixture was trapped in a known amount of standard acid (one-tenth normal H_2SO_4), which was then titrated with a standard base (one-tenth normal NaOH), using methyl orange as an indicator. The decrease in acid is equivalent

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to the amount of volatile base in the intestinal contents. In our study the percentage of volatile base is calculated on the basis of ammonia equivalent to the decrease of standard acid, although actually this is only about 50 per cent ammonia.

The ammonia was determined by thoroughly mixing the distillate with Permutit, decanting the supernatant solution, and washing the Permutit with distilled water. Alkalized water (NaOH) was then added to release the ammonia, the solution was Nesslerized and compared colorimetrically with a standard ammonia solution similarly Nesslerized. Qualitative tests for primary, secondary, and tertiary amines were made on the ammonia free portion by the method described by Hinburg.[11]

Hydrogen ion concentrations were determined with a Leeds-Northrup quinhydrone electrode.

Distillates for injection into normal dogs were made by adding magnesium oxide to intestinal contents of obstructed dogs and employing steam distillation. In this way the necessity of trapping the distillate, as was described above in the analysis of the contents, was obviated.

The volatile base which developed in the fluid contents of the small bowel was investigated by four types of experiments.

In Group A simple mechanical obstruction was produced in 8 dogs by dividing the intestine at the ileocecal junction and inverting the ends. The animals were given a mixed diet before and after the operation. At intervals of from three to nine days they were killed and the intestinal fluid was removed for study.

TABLE I

GROUP A.—SIMPLE ILEAL OBSTRUCTION

ANIMAL NO.	WEIGHT POUNDS	FLUID CONTENT OF BOWEL CONTENT	VOLATILE BASE PERCENTAGE	pH	DURATION OF EXPERIMENT DAYS	CONDITION OF ANIMAL
22	55	1198	0.416	–	3	Good
24	26	580	0.34	–	9	Fair
32	32	–	1.23	–	6	Dead
34	34	200	1.60	–	7	Good
35	36	400	1.45	–	5	Poor
36	32	750	1.61	7.3	6	Good
101	35	–	1.30	7.8	5	Poor
42	42	–	0.14	7.5	5	Good

In Group B long closed loop (127 to 178 cm.) obstructions were studied in 4 dogs. The obstruction was done in two stages, first leaving the distal end of the loop open as a blind enterostomy; and at the second stage 50 to 100 c.c. of intestinal contents taken from an ileostomy, performed on a normal dog, were injected into the loop, after which the obstruction was completed.

TABLE II

GROUP B.—CLOSED LOOP OBSTRUCTION OF SMALL INTESTINE

ANIMAL NO.	WEIGHT POUNDS	FLUID CONTENT OF BOWEL CONTENT	VOLATILE BASE PERCENTAGE	pH	DURATION OF EXPERIMENT DAYS	CONDITION OF ANIMAL
19	55	225	0.38	–	3	Good
27	37	–	1.0	–	2.8	Dead
28	55	130	0.378	–	6	Poor
38	42	1050	0.254	–	3	Good

EFFECTS OF A PROTEIN DIET

Many writers are of the opinion that a diet consisting only of meat causes early death in intestinal obstruction. On account of this belief, Group C dogs (7) were given a pure meat diet before operation and 2 dogs were given a relatively meat-free diet. They were autopsied immediately following death and the fluid contents were analyzed.

TABLE III

GROUP C.—EFFECTS OF PROTEIN DIET ON SIMPLE ILEAL OBSTRUCTION

ANIMAL NO.	WEIGHT POUNDS	DURATION OF LIFE DAYS	DIET*	FLUID CONTENT OF BOWEL CONTENT	VOLATILE BASE PERCENTAGE	NH_3 PERCENTAGE	pH
118	44	3	Meat	200	0.43	0.212	7.9
119	55	3	Meat	400	0.476	0.288	–
122	52	3	Meat	–	–	–	7.8
123	36	1.25	Meat	250	0.43	0.24	–
124	50	2.5	Meat	700	0.325	0.18	7.6
126	56	3	Meat	700	0.36	–	–
110	48	3.75	Meat post-operatively	300	0.519	–	–
121	56	5	No meat	700	0.2	0.11	–
117	44	24	No meat	1700	0.082	0.044	–

*Diet was given for three days previous to operation and continued until death of the experimental animal.

TOXIC EFFECTS OF THE VOLATILE BASES

Since some authors uphold the toxic theory, it was thought advisable to test the toxicity of the distillate containing the volatile bases, as compared to the volatile base free residue (Table V). The alkalinity of the distillate was so high that it was necessary to lower it, by the addition of hydrochloric acid, to about a pH of 8 which is the level occurring at death in simple mechanical obstruction. In 3 dogs the standardized distillate was injected into long closed loops extending from the duodenum to the ileum. In order to obviate the tension factor in the loop, 3 dogs which had esophogostomies with closure of the distal end were obstructed at the ileocecal junction and the distillate was injected into the closed intestinal loop without force. In Dog 138 a large amount of distillate was continually irrigated through the small intestine, being inserted through a duodenal tube and collected by an ileal tube.

TOXICITY OF THE BASIC RESIDUE

In order to compare the residue with the distillate, the following experiments were done. The residue was prepared by heating the intestinal contents of dogs dying from intestinal obstruction to 100° F. and by then distilling it by partial vacuum. The pH of the residue was standardized so as to give a similar reading as is found in intestinal obstruction at death. In 2 dogs the residue was injected into a closed loop extending from the duodenum to the ileum, and in 2 dogs the closed loop extended from the esophagus to the ileocecal junction.

COMMENTS AND RESULTS

It is generally conceded that ammonia, amines, etc., produced in the intestine are products of protein putrefaction. Mathews[7] and Macleod[8] showed that ammonia is split from the protein during digestion and absorption. A part arose from the acid amide as a result of the putrefactive action of bacteria. Still another part was produced by the partial deaminization of the amino-acids during the process of absorption. In addition Mathews pointed out that a large amount of ammonia occurred in the pancreatic secretions as well as in the gastric and intestinal mucosa (40 mg. per 100 gm. of fluid and 36 mg. per 100 gm. of tissue respectively). In all probability the volatile bases as a group originate in this manner.

Cholopoff[2] showed that normal arterial blood contains 0.01 to 0.02 mg. of ammonia per 100 c.c. of blood. In its passage through the wall of the large intestine and also at times through the wall of the small intestine, the blood absorbs ammonia, more with a meat diet and less with a carbohydrate diet, so that the ammonia content in the portal vein after a meat diet may rise to 0.06 mg. per 100 c.c. In the liver a good part of this absorbed ammonia is removed, the hepatic vein showing less ammonia than the portal vein.*

QUANTITATIVE DETERMINATION OF THE VOLATILE BASES

The results given in Table I, Group A, show that the volatile bases occurred in the fluid contents in association with low ileal obstruction, in amounts varying from 0.14 per cent to 1.60 per cent. There appeared to be no relationship between the duration of the obstruction and the concentration of the volatile bases. Likewise the concentration of the volatile bases per se had little influence on the condition of the experimental animal. Similar results were observed in the long closed loop obstructions (Table II, Group B). This group showed no correlation between the percentage of volatile bases, the duration of obstruction, or the condition of the experimental animal.

*Ingraham and Visscher[6] noted accumulation of ammonia in a segment of a normal dog's gut while chloride was being absorbed from it.

INFLUENCE OF PROTEIN DIET

Since Whipple[12] and his coworkers have demonstrated that there is an increased destruction of protein in intestinal obstruction, a preponderance of volatile bases would be expected to occur in animals fed on a meat diet. However, these experiments (Group C) show the concentration to be only slightly greater than that observed in dogs receiving an ordinary diet (Groups A and B), although much greater than in two animals fed a relatively protein-free diet (Nos. 121, 117).

In this group the fluid contents were further analyzed for ammonia, primary, secondary, and tertiary amines as previously described. The results showed that the percentage of ammonia consistently amounted to about one-half of the volatile basic group. The remaining one-half gave positive qualitative tests for primary and tertiary, but negative reactions for secondary amines.

One of the important deductions to be derived from this group is that the animals not only showed earlier signs of toxicity, but that their length of life was greatly shortened. The average was three days as compared to seven days in dogs fed ordinary diets. One dog receiving a relatively meat-free diet lived twenty-four days.

It appears, therefore, that the end-products of protein putrefaction, if not primarily responsible for the cause of death in low ileal obstruction, certainly accelerate a fatal outcome. If the former instance were true, it may be concluded that when the percentage of volatile bases reached a certain fixed concentration, death occurred. Moreover, the high percentage of volatile bases found after death or when the fluid contents were allowed to stand, showed that the chemical action did not stop when it reached the antemortem level. But inasmuch as some of the dogs, with average volatile base levels, were in excellent condition when sacrificed, some other factor must serve in combination with the volatile bases.

SIGNIFICANCE OF THE H-ION CONCENTRATION OF THE FLUID CONTENTS

It has previously been shown[5] that, in contrast to the acid reaction of normal intestinal contents as described by McClendon, Shedlow, and Karpman,[10] Thompson,[9] Grayzel and Miller,[4] Graham and Emery[3] the contents of the obstructed intestine were alkaline. It was also demonstrated that the alkalinity of the fluid contents increased in proportion to the duration of the obstruction until it reached its maximum, a pH of about 8, just before death occurred. After death or if the contents were allowed to stand, the pH decreased rapidly to the acid side.

Wilkie[13] has injected the intestinal contents, obtained from dogs with obstruction, into the bowel of a normal dog without ill effects. Also others[1] have demonstrated that toxic symptoms do not develop when the contents obtained from animals with intestinal obstruction were placed in the bowel of normal animals. The outcome would have been different

probably, if these investigators had taken into consideration not only the pH of the contents, but also the resistance that a normal dog would present as compared to a dehydrated dog with a seven-day obstructed intestine. In fact, a normal dog possesses the ability to lower the pH of a foreign intestinal fluid rapidly. This is shown in the following series of experiments, in which the toxicity of the intestinal contents obtained from dogs dying from intestinal obstruction was tested after the pH had been raised to an antemortem level. The length of life following injection in Dogs 50 and 130 was six and ten hours respectively. In Dog 50 the pH had dropped to 7.1 at death. In Dog 120, 300 c.c. of contents with pH raised to 7.8 was injected into a closed loop. For nine hours following the anesthesia, the dog was unable to stand and twitching of his tongue and neck was noticed. At twenty-four hours he would walk with a staggering gait and was extremely weak. From this time on he became worse, probably the result of the high obstruction. He was sacrificed at 50 hours and the pH of the intestinal fluid contents was 7.6.

TOXICITY OF NH_3 AND VOLATILE BASIC GROUP

In order to test the toxicity of the volatile bases a synthetic solution was used for injection into normal dogs. Since ammonia constituted about 50 per cent of the volatile basic group, it was prepared in a 1.75 per cent solution [$NH_4H\ CO_3$ and $(NH_4)_2\ S$] with a pH of 8.2 and injected into closed loops of otherwise normal dogs. After injection, the animals died in a very short time. A synthetic ammonia solution with a pH of less than 7 had no apparent untoward effect upon the experimental animals. In two experiments distilled water with the pH raised to 8.4 and to 9, with NaOH, then saturated with hydrogen sulphide and injected as before showed no ill-effects twenty and twenty-four hours later, respectively. By employing a solution containing the primary and tertiary amines in addition to ammonia, similar results would be expected since their toxicity increases in proportion to their complexity.

These results would suggest that the end-products of protein putrefaction were toxic only if the pH increased to a certain level. Since the pH influences the dissociation and solubility of ammonia salts, the

TABLE IV

GROUP D.—TOXICITY OF VOLATILE BASE FREE RESIDUE

ANIMAL NO.	WEIGHT POUNDS	RESIDUE INJECTED INTO CLOSED LOOP			LENGTH OF LOOP	DURATION OF EXPERIMENT HOURS	pH AT DEATH
		AMOUNT C.C.	pH	VOLATILE BASE PER CENT			
134	30	500	9.0	0.038	Duodenum to Lower Ileum	26*	8.4
136	34	600	9.0	0.038	Esophagus to Lower Ileum	48*	7.8

*Living and well when sacrificed.

toxicity occurring with a high pH was probably due to the liberation of ammonia, rather than to a direct action upon the intestine.

The results given in Table IV show quite conclusively that, when the distillate containing the volatile bases was removed from the intestinal fluid contents from the dogs dying with intestinal obstruction, the remaining portion did not elicit signs of toxicity when placed in intestinal loops of normal dogs. The pH of the residue was raised to a pH of 9 previous to injection in both experiments. When autopsied twenty-six and forty-eight hours later, the pH had dropped to 8.4 and 7.8 respectively.

The results obtained with the volatile basic distillate (Table V) were quite different. They showed that when the pH was set at a fixed level, the injected distillate caused early death in all animals.

TABLE V

GROUP E.—TOXICITY OF DISTILLATE CONTAINING VOLATILE BASES

ANIMAL NO.	WEIGHT POUNDS	DISTILLATE INJECTED INTO CLOSED LOOP			LENGTH OF LOOP	DURATION AT DEATH HOURS	DEATH pH
		AMOUNT C.C.	pH	VOLATILE BASE PER CENT			
113	20	450	9+	0.485	Duodenum to lower ileum	5	-
114	45	400	9+	0.48	Duodenum to lower ileum	9	-
117	42	400	9+	0.37	Duodenum to lower ileum	4.5	8.8
128a*	24	800	8.2	0.43	Esophagus to lower ileum	8 to 14	-
127†	36	800	9.0	0.43	Esophagus to lower ileum	3	-
135	28	500	About 8.6	0.43	Esophagus to lower ileum	24	-
138‡	48	1000	8.4	0.43	Duodenum to lower ileum	16	-

*Blood CO_2 was 58 three or four hours following injection.

†Hydrogen ion concentration of urine was 7.1 just before death.

‡1,000 c.c. of the distillate was continuously irrigated through small intestine, being injected into a duodenal tube and collected by an ileal tube.

It must be concluded, therefore, that if the volatile bases of the fluid intestinal contents in low ileal obstruction occur in a certain concentration, death will occur, providing the H-ion concentration is at a certain level. This does not signify, however, that they are the primary cause of death in clinical intestinal obstruction.

SUMMARY AND CONCLUSION

An experimental study of the volatile bases developing in the fluid contents of the intestine following low ileal mechanical obstruction has been made. They were found to occur in concentrations varying from 0.14 per cent to 1.60 per cent. There appeared to be no correlation between the concentration of volatile bases, the duration of obstruction, or the condition of the experimental animal.

The volatile bases were further analyzed for ammonia, primary, secondary, and tertiary amines. Ammonia consistently amounted to about 50 per cent of the group. The remaining half gave positive qualitative tests for primary and tertiary but negative reactions for secondary amines.

Although the volatile bases are products of protein putrefaction, a pure meat diet only slightly increased their concentration in the fluid contents of the obstructed intestine. The length of life in dogs obstructed at the ileocecal junction was greatly shortened when they were fed a pure meat diet in contrast to an ordinary or a meat-free diet.

When the pH of the intestinal contents obtained from dogs dying from intestinal obstruction was raised to an antemortem level and placed into bowel loops of normal animals, toxic symptoms appeared soon, followed by death of the experimental animals. Likewise, the distillate containing the volatile bases was found to possess the same characteristics.

In contradistinction to the toxic effects of the distillate, it has been shown that the action of the volatile base-free residue is entirely innocuous.

This does not necessarily signify that the volatile bases are the primary causes of death in intestinal obstruction, but it does mean that if they are present in a high enough concentration at an optimum pH and if they are absorbed, severe toxic symptoms will occur followed by a fatal outcome.

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ACUTE INTESTINAL OBSTRUCTION

I. The Rôle of Bacteria in Closed Jejunal Loops

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OF THE various theories proposed to explain the cause of death in intestinal obstruction, two have received the most attention, viz.: (1) the loss of large amounts of fluids from the upper part of the gastrointestinal tract by vomiting; and (2) the absorption of some toxic substance formed in the obstructed bowel.

The serious consequences resulting from the loss of a large amount of fluid from the upper intestinal tract by vomiting have been repeatedly observed in both the experimental laboratory and in patients with acute bowel obstruction. In the experimental animal symptoms can be greatly altered and the length of life following acute obstruction definitely prolonged by the parenteral administration of adequate amounts of isotonic salt solution. Death, however, although delayed, is never prevented, and many workers feel that it is the absorption of some toxic material from the obstructed bowel which finally proves fatal.

Murphy and Brooks[1] and Dragstedt[2] found that closed intestinal loops were compatible with life if the bacteria in the loops were eliminated by adequate drainage before closure. If bacteria and tissue necrosis were present in the bowel segments, the animals died.

Williams,[3] in 1926, advanced the hypothesis that the Welch bacillus was the organism primarily responsible for the toxemia in intestinal obstruction. This conclusion was based on animl experiments carried out by methods which seem to us to be unsatisfactory and will be discussed later. Later workers have not been in agreement on this point.

McIver and others,[4] Oughterson and Powers,[5] and Thurston[6] were unable to find sufficient evidence that *Cl. welchii* was the important factor in acute intestinal obstruction, while the observations of Bower and Clark[7] as well as experiments by Morton and Stabins[8] seemed to show that *Cl. welchii* toxin played some part in the symptoms.

In the present experiments a study was made of the bacteriology and toxicity of the contents of obstructed bowel segments.

METHODS

Closed loops of upper jejunum were made in dogs under ether anesthesia. A portion of jejunum, varying from 15 to 30 cm. in length,

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was sectioned at either end and the ends closed by purse-string sutures. The continuity of the bowel was reestablished by either a side-to-side or an end-to-end anastomosis. Following operation, the animals were placed in cages and given only water by mouth. When the animals appeared to be critically ill, usually thirty-six to seventy-two hours after operation, a second laparotomy was done and the obstructed loop removed. The fluid from this excised loop was immediately placed in sterile containers and used for bacteriologic examination and toxicity studies.

The loop contents were centrifugalized at high speed for forty-five minutes and the supernatant fluid carefully removed and used for mouse inoculations. Mice weighing from 17 to 20 gm. were given from 0.2 c.c. to 0.5 c.c. of fluid intravenously, control animals being given the same quantity together with 0.05 *Cl. welchii* antitoxin. Deaths of mice occurring within forty-eight hours only were recorded.

The following technique for culturing the loop fluid was selected because it seemed the best procedure for isolating as many types of bacteria as possible and for simultaneously determining quantitative relationships. Decimal dilutions of the loop fluid were made in tubes of sterile saline, from which lactose broth, Robertson's beef heart medium, and freshly boiled milk were inoculated.

Lactose broth was used to detect the presence of the coli-aerogenes group of bacteria. All lactose broth cultures giving the reaction of *B. coli* were streaked out on eosin-methylene blue agar plates for final identification. Beef heart medium was used because it satisfied the growth requirements of the gram-negative intestinal bacteria, the cocci, and many of the sporulating and nonsporulating anaerobes. The cultures were incubated one week. The highest dilutions showing growth in beef heart medium were used for inoculating aerobic and anaerobic blood agar plates. Identification of the predominating types of bacteria was made from these.

Milk was used for the estimation of the numbers of *Cl. welchii*. The inoculum was heated at 80° C. for ten minutes. The cultures were incubated in an anaerobic jar, and when typical stormy fermentation occurred, they were removed from the jar and blood agar plates were streaked for the isolation of *Cl. welchii*. This method gives only an estimate of the number of spores present; the number of vegetative forms, perhaps, is much greater.

The 1:10 dilution of loop fluid was used for direct inoculation of blood agar and eosin-methylene blue agar plates. Blood agar plates were incubated anaerobically at a temperature of 37° C. for four days. Aerobic blood agar and eosin-methylene blue plates were incubated for twenty-four hours. Identification of the predominating organisms on these plates was then made.

A strain of *Cl welchii* was isolated from each animal. Identification of the organism was established by the characteristic stormy fermentation of milk, typical colonies on blood agar, and specific toxin production. The strains isolated were grown on chopped veal-beef infusion medium for toxin production in accordance with the procedure used by Bengston[9] at the National Institute of Health at Washington, D. C. It was necessary to transplant from a very young beef heart culture and incubate the culture for not longer than from sixteen to twenty-two hours to produce a potent toxin. The toxin was centrifugalized and filtered through a Berkefeld "N" candle before being used for injection. In each case the filtrate was cultured and found to be sterile. One group of mice was injected intravenously with 0.5 c.c. of this filtrate, and another group of animals was given 0.05 c.c. of *Cl. Welchii* antitoxin, previously mixed with the supernatant fluid and allowed to stand at room temperature for forty-five minutes.

RESULTS

The results of the bacteriologic studies are presented in Table I. *Cl. welchii,* with one exception, was present in large numbers. The actual number of vegetative forms was probably much greater than

TABLE I

PREDOMINATING ORGANISMS IN OBSTRUCTED LOOP FLUID*

DOG	AEROBES	ANAEROBES
177	*B. aerogenes* 10^{-9}	*Cl. welchii* 10^{-4}
	B. coli 10^{-9}	
181	*B. coli* 10^{-11}	*Cl. welchii* 10^{-4}
	Fluid aspirated after 23 hours	
489	None	*Cl. welchii* 10^{-7}
	Fluid aspirated after 97 hours	
	None	*Cl. welchii* 10^{-6}
		Nonhem. gram + rods 10^{-8}
512	*B. coli* 10^{-8}	*Cl. welchii* 10^{-6}
261	*B. coli* 10^{-11}	*Cl. welchii* 10^{-7}
390	*B. coli* 10^{-11}	*Cl. welchii* 10^{-7}
	B. proteus	Nonsporulating gram-rods
493	*B. coli* 10^{-12}	*Cl. welchii* 10^{-6}
526	*B. coli* 10^{-10}	Gram + bacilli (not welchii)
422	*B. coli* 10^{-11}	*Cl. welchii* 10^{-2}

*Decimal dilutions expressed as exponents.

those recorded, since the method used detected only the number of spores. *B. coli* was usually present in large numbers also. Occasionally *B. aerogenes* and *B. proteus* were found, but in the majority of cases, *Cl. welchii* and *B. coli* completely predominated the flora. In one case, the Welch bacillus was the only organism isolated. Few anaerobic organisms other than *Cl welchii* were ever encountered, although a search was made for these. The cocci were also conspicuously absent.

TABLE II

EVIDENCE FOR THE PRESENCE OF *Cl. welchii* TOXIN IN THE OBSTRUCTED LOOP FLUID. TOXIN PRODUCING CAPACITY IN VITRO OF *Cl. welchii* ORGANISMS ISOLATED FROM THE OBSTRUCTED LOOP CONTENT

DOG	RESULTS OF INOCULATION WITH LOOP FLUID				IN VITRO PRODUCTION OF TOXIN BY HOMOLOGOUS STAINS	
	TEST MICE		ANTITOXIN CONTROLS		TEST MICE*	
	NO. OF DEATHS	NO. OF SURVIVALS	NO. OF DEATHS	NO. OF SURVIVALS	NO. OF DEATHS	NO. OF SURVIVALS
177	0	5	1	4	2	1
181	4	1	1	4	3	0
	Fluid aspirated after 23 hours					
489	0	5	0	4	3	0
	Fluid aspirated after 97 hours (end of experiment)					
	0	4	0	5		
512	1	1	1	0	2	0
261	1	1	2	0	3	0
390	4	0	2	3	No toxin demonstrated	
493	3	1	3	0	No toxin demonstrated	
526	3	1	2	1	*Cl. welchii* not found	

*Antitoxin controls all survived.

The results of the toxicity studies are presented in Table II. No conclusive evidence of the presence of *Cl. welchii* toxin in the loop fluid was found. The method for preparing the fluid for mouse injection is open to criticism in that the fluid was not sterile. Prolonged filtration, which is necessary with this type of material, and heating may be destructive to *Cl. welchii* toxin, and for that reason were avoided. However, the survival of the mice in some of these experiments is evidence against the presence of welchii toxin. If the fluid contained a toxic entity, such as *C. welchii* toxin, one would expect uniform deaths among the animals injected in a short period of time. This was not the case in these experiments.

The strains of the Welch bacillus isolated from each loop, with two exceptions, were good toxin producers when grown in suitable medium. In one experiment none of the mice receiving loop fluid died. This fluid, however, contained one of the best toxin-producing strains which was encountered.

In two instances (Dogs 181 and 489) blood was drawn three weeks after obstruction to test for the presence of *Cl. welchii* antitoxin. Mice injected with 0.5 c.c. of filtrate (prepared with homologous strains) mixed with 0.1 c.c. of the blood serum of the dog and allowed to stand at room temperature for forty-five minutes before injection, all died.

Bull and Pritchett[10] have shown that young cultures of *Cl. welchii* contain the maximum of toxin, and that with prolonged incubation, the toxicity decreases. With this idea in mind, fluid was aspirated from an intestinal loop (Dog 489) twenty-three hours after obstruction and tested for toxin. None of the mice receiving the fluid died.

DISCUSSION

Williams mixed the contents of the small intestine with an equal amount of ice cold saline solution and passed this through a Berkefeld candle in an atmosphere of hydrogen in the ice chest. The filtration always took from eighteen to twenty-four hours. Mice were injected intramuscularly, using 0.75 c.c. of filtrate plus 0.25 c.c. of normal horse serum in one series, and 0.75 c.c. of filtrate together with 0.25 c.c. of *Cl. welchii* antitoxin in another series. Striking results were tabulated, showing large numbers of deaths in the first group and no fatalities in the protected group. The mice were kept under observation for six days. All the mice which died were examined for the possibility of infection with *Cl welchii,* as opposed to toxemia, by heart blood cultures taken as soon after death as possible. He states that all these cultures were negative as regards *Cl. welchii,* although occasionally streptococci and coliforms were found. Assuming that examination was made soon enough after death to prevent the possibility of postmortem invasion of organisms, his results indicate that the filtrates were not sterile.

It seems logical that the intramuscular injection of quantities as large as 1 c.c. into a mouse could produce enough tissue injury to afford conditions suitable for the elaboration of a toxin by the Welch bacillus, if that organism were contained in the fluid injected. The fact that the filtrates Williams used were not sterile seems to indicate that *Cl. welchii* may have been present in the filtrates. Despite his failure to demonstrate *Cl. welchii* in the heart blood, the organisms would, more likely, have localized at the site of injection. It is possible that the mice died from a toxin elaborated by *Cl. welchii* in the tissues rather than from any toxin contained in the loop fluid. Williams kept the mice under observation for six days. Our experience, and that of others[9, 10] has been that intravenous injection of a potent toxin of *Cl. welchii* in quantities much less than 1 c.c. will kill a mouse in from two to forty-eight hours. Death of mice after forty-eight hours may be attributed to other causes.

McIver and others are of the opinion that the strains of *Cl. welchii* found in the intestinal tract are of a low degree of virulence and are not good toxin producers. The strains studied here, with two exceptions (Dogs 390 and 493), all produced toxin when grown in a suitable medium for from sixteen to twenty-four hours. It was found that the use of old cultures for inoculation of the chopped veal medium gave poor results, and that the time of incubation of cultures for the production of toxin was important and varied considerably.

Wilsdon[11] has recently divided the *Cl. welchii* group into four types, each differing in the antigenic structure of the toxin produced. Types A, B, and C produced the maximum of toxin within twenty-four hours. However, Type D, consisting of a series of strains of animal origin, pro-

duced a toxin which did not reach its maximum for three to five days. This may explain our failure to obtain toxins with strains isolated from Dogs 390 and 493.

SUMMARY AND CONCLUSIONS

Bacteriologic studies were made on the fluid obtained from closed intestinal loops in eight dogs.

Cl. welchii and *B. coli* were found to be the predominating organisms. In most instances the Welch organisms were shown to be capable of producing a potent toxin in vitro. No definite evidence of *Cl. welchii* toxin in the loop fluid was demonstrated.

The present study does not substantiate the view that the toxemia of acute intestinal obstruction is due to the specific toxin of the Welch bacillus.

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ACUTE INTESTINAL OBSTRUCTION

II. The Permeability of Obstructed Bowel Segments of Dogs to *Clostridium botulinum* Toxin

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THE rôle played by bacteria and their products in intestinal obstruction is an unsettled problem. This subject has been discussed in a previous report. The permeability of the obstructed bowel has been studied by other workers with regard to some of the chemical poisons, but no satisfactory method has been devised to study the permeability to bacterial products. Since the dog was known to be quite resistant to oral administration of botulinum toxin and since this toxin is of bacterial origin, it was thought that this substance might better meet the conditions found in intestinal obstruction. Furthermore, this is one of the most potent bacterial toxins and can be detected in very small quantities.

Cultures of *Cl. botulinum* Type A were grown in Kjeldahl flasks containing Robertson's beef heart medium under a vaseline-paraffin seal. These were incubated at 37° C. for from four to fourteen days, at the end of which time the supernatant fluid was centrifugalized at high speed for one-half hour. The resulting supernatant fluid was pipetted off carefully and constituted the toxin used in these experiments. The potency of the toxin was determined by intraperitoneal injections of 1 c.c. of decimal dilutions into each of two mice, weighing from 17 to 20 grams each. The mice were kept on test for forty-eight hours. The same procedure was used for detection of toxin in the blood serum of dogs. Control mice were inoculated with 0.25 c.c. of *Cl. botulinum* antitoxin.

I. PERMEABILITY OF NORMAL BOWEL SEGMENTS

To determine whether the normal dog intestine was permeable to *Cl. botulinum* toxin, 20 dogs were fed varying quantities of highly potent botulinum toxin* with a stomach tube. Four dogs fed 25 c.c. and 4 dogs fed 50 c.c. gave no evidence that absorption was obtained. A variation in results occurred when 100 c.c. quantities were fed. In 2 out of 12 dogs fed from 85 to 100 c.c., there was no absorption of toxin.

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*Titrations of toxin used in feeding experiments were comparable with those used in later obstruction experiments.

Absorption was questionable in 4 other animals fed 100 c.c. (antitoxin controls died in 2 of these). When 2 other animals were fed, considerable bleeding about the mouth occurred, and this is thought to have been responsible for the early death of the dog and ready absorption of toxin. In subsequent feedings, the dogs were put under light ether anesthesia. This eliminated struggling and avoided abrasions in the oral mucosa.

In 4 other dogs fed 100 c.c. of potent toxin, absorption was demonstrated, but in only 1 of these was it sufficient to cause the death of the animal (died nine days after feeding). Furthermore, the toxin in these 4 animals was never absorbed in quantities sufficient to be detected by mouse injection in a 1:10 dilution of the serum. However, toxin was sometimes demonstrated in this dilution in the experiments with acute obstruction of loops of the small intestine.

II. ABSORPTION OF *Cl. Botulinum* TOXIN FROM THE PERITONEAL CAVITY OF DOGS

Dogs were given intraperitoneal inoculations of toxin to determine the time of appearance in the blood and to gain some quantitative relationship between potency of toxin and amount absorbed. Results are recorded in Table I. Two dogs, Cases 2 and 3, were inoculated with 3 c.c.

TABLE I

BOTULINUM TOXIN IN BLOOD SERUM OF DOGS INJECTED INTRAPERITONEALLY WITH 3 C.C. OF TOXIN

DOG	M.L.D. OF TOXIN	DILUTION OF SERUM	TOXIN IN BLOOD SERUM					RESULTS OUTCOME FOR DOG
			2 HR.	4-4½ HR.	6-6½ HR.	12 HR.	24 HR.	
1	10^{-4}+	0			++	++	++	Fatal
	*	1:10			++	++	++	
2	10^{-3}+	0	--	--	--			Survived
	10^{-4}-	1:10	--	--	--			
3	10^{-3}+	0	--	--	--			Survived
	10^{-4}-	1:10	--	--	--			
4	2×10^{-4}+	0	--	--	--			Survived
	10^{-4}-	1:10	--	--	--			
5	2×10^{-4}+	0	++	++	++			Fatal
	10^{-4}-	1:10	--	+-	++			
6	10^{-4}+	0	++	++	++			Fatal
	*	1:10	++	++	++			
		1:100		+-	+-			
7	10^{-4}	0	++	++	++			Fatal
	*	1:10	++	++	++			
		1:100		--	+-			

+ denotes death of 1 mouse; - denotes survival of 1 mouse; *, not tested in a dilution greater than 10^{-4}; blank space, no test made.

of filtered toxin with the M. L. D.* varying between 10^{-3} to 10^{-4}. No toxin was demonstrated in the blood serum of these two animals, and they survived with no ill effects. In Case 1 where a nonfiltered toxin

*M. L. D. determined roughly by decimal dilutions of toxin injected intraperitoneally into white mice.

with an M.L.D. of at least 10^{-4} was used, the dog died and toxin was demonstrated in the blood serum in six hours (no titrations were made earlier). Further experiments demonstrated that when 3 c.c. of potent toxin is administered intraperitoneally, the toxin enters the blood within two hours, increases in amount with time, and is fatal to the dog. The negative results obtained in Case 4 may possibly be explained by the toxin entering a viscus instead of the peritoneum. The same toxin was used in Case 5 with fatal results.

III. ACUTE INTESTINAL OBSTRUCTION EXPERIMENTS

Dogs were prepared with isolated loops of small intestine, the ends carefully infolded by means of purse-string sutures, and the continuity of the intestinal tract reestablished by means of end-to-end anastomoses. *Cl. botulinum* toxin was inoculated into the loop with a fine needle, care being taken to prevent any toxin from entering the peritoneal cavity. The loop was put back into the peritoneum and the incision closed. Blood was drawn at intervals and the serum inoculated into white mice to test for the presence of *Cl. botulinum* toxin. The results are presented in Table II.

TABLE II

BOTULINUM TOXIN IN BLOOD SERUM OF DOGS RECEIVING 10 C.C. OF TOXIN INTO OBSTRUCTED LOOPS OF SMALL INTESTINE

DOG	M.L.D. OF TOXIN	DILUTION OF SERUM	TOXIN IN BLOOD SERUM					
			3-6 HR.	16-18 HR.	20-24 HR.	30 HR.		HEART BLOOD AT DEATH
Acute Obstruction Experiments								
8	10^{-4}+	0		−−				++
	10^{-5}−	1:10		−−				−− (36 hr.)
9	10^{-3}+	0	−−	−−		−−	Experiment	
	10^{-4}−	1:10	−−	−−		−−	Discontinued	
10	2×10^{-4}+	0	−−	−−	+			++
	10^{-4}−	1:10	−−	−−				++ (60 hr.)
Obstructed Loops Previously Drained								
11	10^{-3}+	0	−−		−−	−−	++	++
	10^{-4}−	1:10	−−		−−	−− (40 hr.)	++ (64½ hr.)	++ (69½ hr.)
12	10^{-3}+	0	−−		−−	−−	−−	++
	10^{-4}−	1:10	−−		−−	−−	−−	++
		1:50				(40 hr.)	(63¾ hr.)	++
Obstructed Loop Previously Washed with Tannic Acid Toxin Inoculated After Obstruction Began								
13	10^{-4}+	0	++	++	++			
	10^{-5}−	1:10	−−	+−	++			
		1:100	−−	−−	++			

+ denotes death of 1 mouse; − denotes survival of 1 mouse; blank space, test not made.

In Case 8 the dog died in thirty-six hours. Toxin was demonstrated in the heart blood serum and in the loop contents. The pathologic picture was not typical of acute obstruction, the loop contained no fluid (solid fecal material only) and was not noticeably distended. This suggests that the death may have been due to factors other than obstruction. The possibility suggests itself that some of the toxin may have leaked from the infolded ends of the loop. Were this the case, one would expect to find toxin in the blood serum at an early stage, if

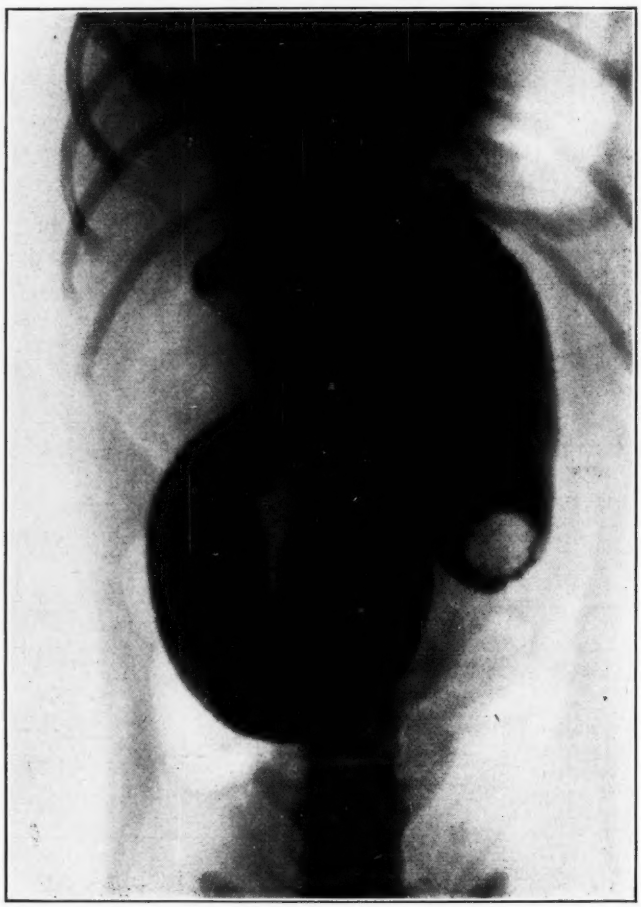

Fig. 1.—Dog 271; photograph of x-ray taken three days after obstruction.

the results from the intraperitoneal inoculations are taken into consideration. No toxin was demonstrated in Case 8 before the death of the animal (thirty-six hours).

In Case 9 the toxin was discovered to be of low titer after the dog had been inoculated. No toxin was demonstrated in the blood serum within thirty hours, and, when the dog became ill, the loop was removed to save the animal. No further titrations were made on the blood serum, but toxin was found in a specimen from the isolated loop.

Dog 10 died in sixty hours. The loop had perforated. This perforation was apparently quite recent since there was still considerable fluid in the loop. Blood drawn twenty-four and a half hours after inoculation contained some toxin as did the heart blood in a 1:10 dilution.

IV. PERMEABILITY OF *Cl. Botulinum* TOXIN FROM ISOLATED LOOPS PREPARED FROM THIRY FISTULAS

Dogs were prepared with isolated loops of jejunum closed at one end, the other open end brought to the outside through a stab wound in the abdominal wall. The loops were permitted to drain for eight months

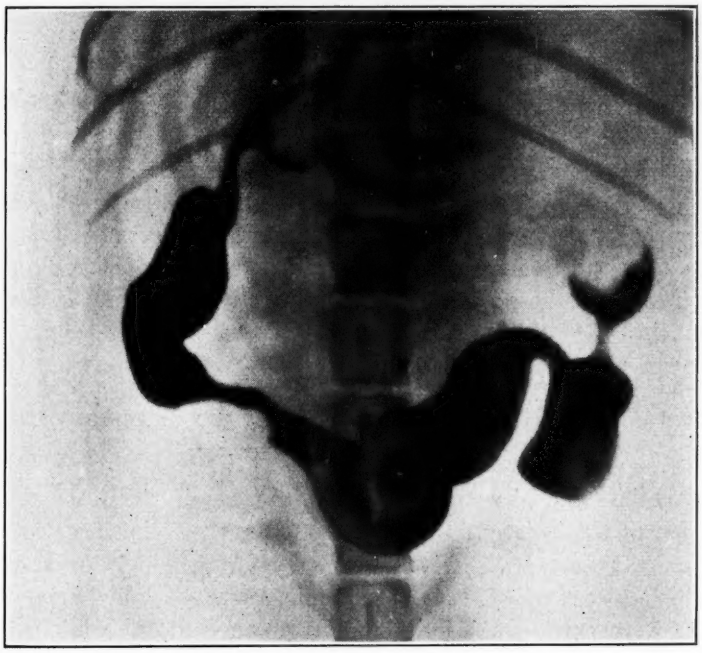

Fig. 2.—Dog 597; photograph of x-ray taken seven days after operation.

and then were carefully closed from within. At this time they appeared much atrophied. In Cases 11 and 12, 10 c.c. of botulinum toxin were injected into the loop after closure. The results are recorded in Table II.

As may be seen from the table, toxin was found in the dog blood in Case 11, a few hours before death. In Case 12 the toxin was found in the heart blood taken shortly after death, but none was found in the serum twenty-three hours before death. Blood cultures were made each time blood was drawn to check on the possibility of septicemia resulting from rupture of the loop. All cultures remained sterile with the exception of one made from blood taken very shortly after death of the animal.

A third dog received no botulinum toxin. Cultures of the loop made before closure showed no growth. The dog died in eight days. A pure culture of hemolytic streptococci was obtained from the loop fluid. This fluid was not toxic for mice.

V. EXPERIMENTS WITH ISOLATED LOOPS OF INTESTINE WASHED WITH TANNIC ACID BEFORE CLOSURE

Two dogs were prepared with isolated loops of jejunum, washed with a 6 per cent solution of tannic acid before closure, the purpose being to minimize secretion and distention before the infolded loops had healed. One dog died from peritonitis, and the second dog was allowed to go for three days, at which time a second laparotomy was performed. The loop was very distended and darkened in a few areas. Two hundred cubic centimeters of brownish fluid were aspirated, and hot packs were applied to the loop until peristalsis had returned and the circulation appeared good with the exception of a few areas opposite the mesenteric attachment. Ten cubic centimeters of botulinum toxin were injected into the loop. The results are tabulated in Table II.

From the one experiment which was carried to completion, it may be seen that the toxin readily passed through the damaged intestinal wall. There was some leakage from the syringe when the toxin was injected, but the amount was calculated as too small to be detected in the general circulation by mouse injection.

VI. RELATIONSHIP OF ABSORPTION OF TOXIN AND DISTENTION OF THE ISOLATED LOOPS OF BOWEL

To correlate the absorption of botulinum toxin with the degree of distention of the isolated intestinal segments, the following experiments were undertaken. Dogs were prepared with isolated loops of ileum into which 10 c.c. of *Cl. botulinum* toxin and 20 c.c. of barium sulfate solution were carefully injected before closure of the abdomen. The animals were x-rayed following the operation, and at the time a blood specimen was drawn for toxin titrations.

X-ray pictures were always made on the same scale and by this procedure, the size of the loop could be followed easily. Contraction due to peristaltic movements sometimes complicated the picture, but, in general, the increase or lack of increase in size could be readily determined. Photographs of two such x-rays are shown in Figs. 1 and 2. Tracings of the x-rays were made and are shown in Figs. 3 to 13. Results of the toxin assays are presented in Table III.

No toxin was demonstrated in the blood serum of 5 of the 11 dogs studied in this manner. The toxin used in one experiment (Dog 316) was found to be of a very low titer, and, although there was a gradual and marked distention of the isolated loop, no toxin could be detected

TABLE III

BOTULINUM TOXIN IN BLOOD SERUM OF DOGS RECEIVING 10 C.C. OF BOTULINUM TOXIN AND 20 C.C. OF BARIUM SULFATE SOLUTION INTO OBSTRUCTED LOOPS OF ILEUM

DOG	M.L.D. OF TOXIN	DILUTION OF SERUM	TOXIN IN BLOOD SERUM AFTER								TOXIN IN LOOP AT DEATH OF DOG*
			1 DAY	2 DAYS	3 DAYS	4 DAYS	5 DAYS	6 DAYS	7 DAYS	8 DAYS (OR MORE)	
316	10^{-1} +	0	- -	- -	- -	- -	- -	- -	- -	- -	
	10^{-2} -	1:10	- -	- -	- -	- -	- -	- -	- -	- -	Not tested
										11 days = =	
338	10^{-4} +	0	- -	- -							
	10^{-5} -	1:10	- +	Death of dog							+
391	10^{-4} +	0	- -	- -							
	10^{-5} -	1:10		- -							Not tested
				Death of dog							
586	10^{-5} +	0	- -	- -							
	10^{-6} -	1:10		Death of dog							Not tested
611	10^{-4} +	0	+ -	Dog died—distemper							
	10^{-5} -	1:10	- -								+
319	10^{-5} +	0	- -	- -	- -		- -	+ After death			Not tested
	10^{-6} -	1:10			- -		- -				
501	10^{-5} +	0	+ +	+ +	Death of dog						
	10^{-6} -	1:10	- -	- -							Not tested
388	10^{-4} +	0	+ -	- -	+ + +	+ +	+ +	Death of dog			+
	10^{-5} -	1:10		- -	- -	+ -	+ +				
271	10^{-5} +	0	- -	- -	+ +	+ +	+ +	- -	+ -		+
	10^{-6} -	1:10	- -	- -	- -	- -	- -	- -	- -		
									Death of dog		
621	10^{-5} +	0	- -	+ +	+ -	+ -	+ +	+ +	+ -	Death of dog	-
	10^{-6} -	1:10	- -	- -	- -	+ +	- -	- -	- -		
597	10^{-5} +	0	- -	- -	- -	+ +	+ +	+ -	+ +	+ +	
	10^{-6} -	1:10	- -	- -	- -	- -	- -	- -	+ -	- -	+
									9 days ± =	Dog killed 12th day	

*Determined by guinea pig feeding experiments.

+ denotes death of 1 mouse; - denotes survival of 1 mouse; blank space, not tested.

in the blood stream. The remaining 4 animals died within forty-eight hours, and in only 1 was there any distention of the isolated loop.

Evidence of absorption was obtained in 6 of the 11 dogs, and this was usually concomitant with the distention of the isolated loops. In the case of Dog 319 no toxin was found in the blood until after the

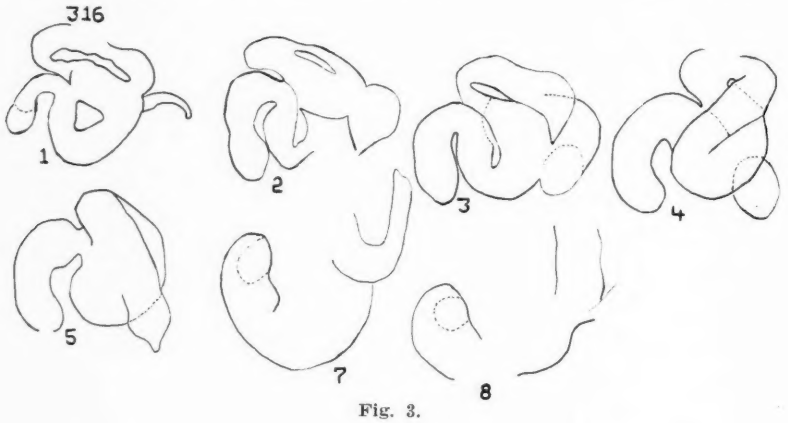

Fig. 3.

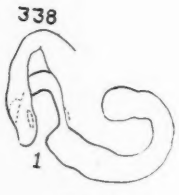

Fig. 4.

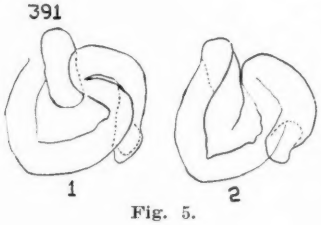

Fig. 5.

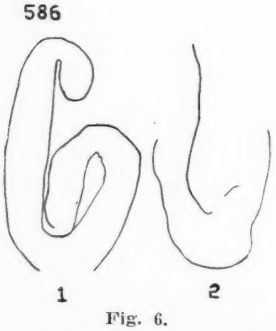

Fig. 6.

Fig. 7.

Figs. 3-13.—Tracings of x-rays. Numbers placed below each loop in Figs. 3 through 13 indicate the number of days after obstruction was produced.

death of the dog, although the loop was shown to have increased in size after forty-eight hours. At autopsy it was found that the loop had ruptured and the absorption of toxin noted at death was, in all probability, directly from the peritoneal cavity.

Dog 501 died in three days. Autopsy revealed a generalized peritonitis and a rupture (apparently quite recent) in the loop which cor-

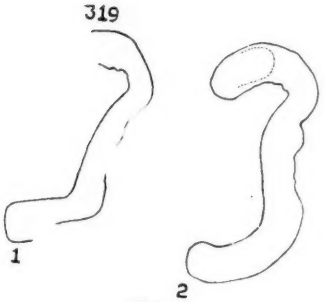

Fig. 8.

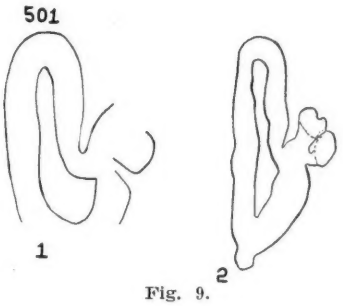

Fig. 9.

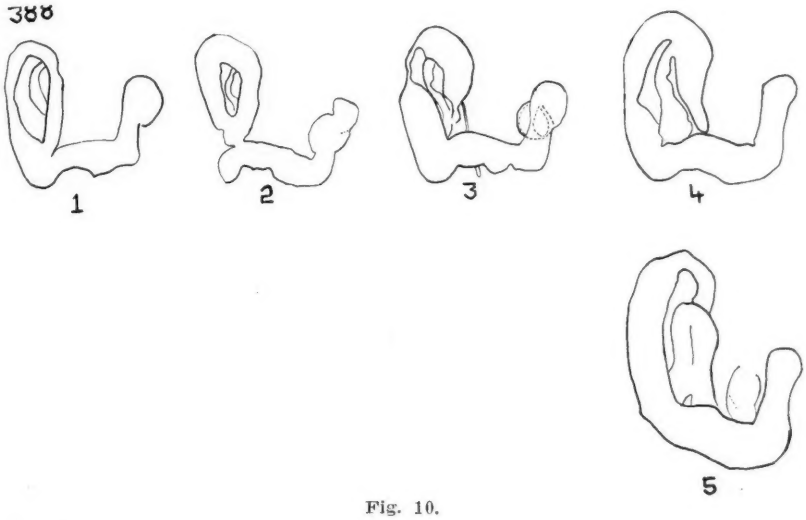

Fig. 10.

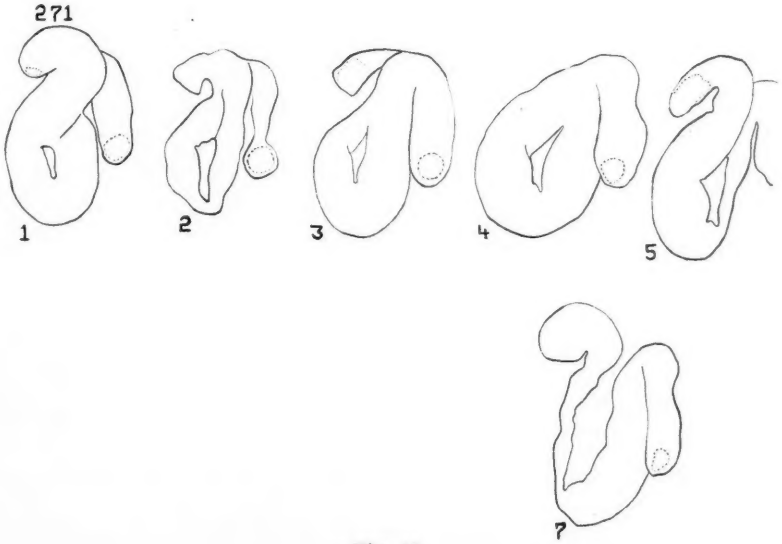

Fig. 11.

Figs. 8-11 —See opposite page for legend.

responded to the outpouching evident in the x-ray taken on the second day after operation. Toxin was found to be present in the blood stream twenty-four and again forty-eight hours after operation.

In 4 of the animals rather clear cut evidence of absorption of toxin into the blood stream was obtained when distention of the loop occurred. In Dog 388 death occurred on the sixth day after operation. X-ray

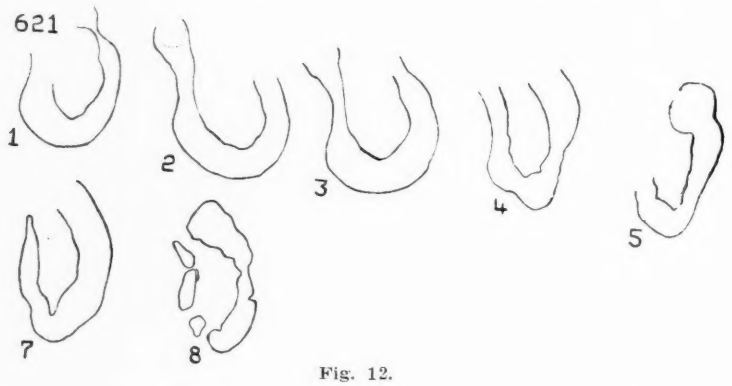

Fig. 12.

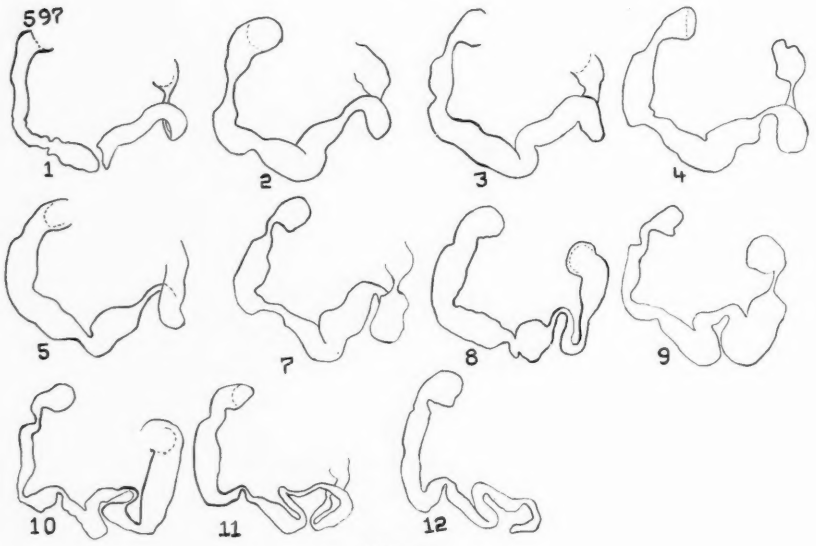

Fig. 13.

Figs. 12 and 13.—See page 346 for legend.

pictures revealed a steady increase in size of the loop each day, and a steady increase of botulinum toxin was found in the serum. It may be noted that in Dog 621, the greatest amount of toxin was found four days after obstruction, after which time it decreased steadily. From the x-ray pictures one may note that the distention was greater on the third day, after which it decreased until the eighth day.

Blood drawn from Dog 271 on the third, fourth, and fifth days after operation contained toxin. The serum was not toxic on the sixth day. Unfortunately no x-ray picture was obtained that day. On the seventh day presence of toxin was questionable, although the x-ray showed the loop to be enormously distended. The quantity of toxin in the serum of Dog 597 appeared to decrease after the seventh day. Pictures of the loop show that it decreased steadily in size after the tenth day.

DISCUSSION

Little is known about the absorption of toxin products from obstructed loops of the bowel. The reason for this lack of knowledge is due to the difficulties of assaying many of the toxic metabolic products formed by the enormous number of bacteria multiplying in the obstructed intestinal tract. The permeability to dyes and to chemical poisons does not offer a parallel situation to what naturally happens. It appeared to us that the toxin of *Cl. botulinum* was particularly well adapted to this type of experimentation, since it is a poison produced by bacteria; it is of sufficient potency so that very small amounts can be detected by the intraperitoneal injection of white mice, some of which have been protected with homologous antitoxin.

Fortunately the dog has considerable resistance to this toxin as evidenced by the fact that 100 c.c. of a potent toxin may be fed to dogs with impunity; whereas, a fraction of 1 c.c. is lethal for man, monkeys, and guinea pigs. This resistance enables the dogs to survive for a long time when a lethal dose of toxin for the white mouse (intraperitoneal route) is present in each cubic centimeter of dog's blood. Another advantage to the use of botulinum toxin was that the toxin is not readily destroyed in the contents of the isolated loop of bowel.

Our experiments indicate that the toxin of *Cl. botulinum*, which in moderate quantities is not absorbed from the normal intestinal tract of dogs, is readily absorbed from obstructed bowel segments. The results strongly indicate that when distention occurs, the toxin enters the blood stream. The possibility that the toxin may have leaked from the loops or may have been absorbed from the loop ends is precluded in view of the fact that it took a long time for it to be detected in the blood when introduced into isolated loops of bowel; whereas, toxin introduced intraperitoneally can be demonstrated in as short a period as two hours.

SUMMARY AND CONCLUSIONS

1. *Cl. botulinum* toxin has not been demonstrated to pass through the normal intestinal wall of dogs when fed in quantities of 50 c.c. or less. Feeding of larger quantities occasionally resulted in absorption, but never in large amounts.

2. This toxin injected intraperitoneally, in suitable doses, is readily absorbed into the blood stream. The toxin may be demonstrated in the blood within two hours; it increases in quantity with time, and is lethal to the dog.

3. Absorption of *Cl. botulinum* toxin placed in small quantities in obstructed segments of the small intestine of dogs has been demonstrated. The distention of the isolated loops was followed by x-ray pictures and there appeared to be some correlation between the demonstration of toxin in the blood stream with distention of the loops. Gross necrosis of the distended segments did not always occur where there was absorption, but devitalization of the bowel segments greatly facilitated the appearance of toxin in the blood stream.

PNEUMOPERITONEUM IN PERFORATIONS OF THE GASTRO-INTESTINAL TRACT

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INTRODUCTION

SINCE the diagnosis of "acute abdomen" is no longer, in all cases, considered to be indicative of an immediate operation, an ever increasing importance must be placed on the correct diagnosis of acute lesions within the peritoneal cavity. That the usual physical examination by means of inspection, palpation, percussion, and auscultation is insufficient to arrive at a correct diagnosis in many such cases is only too well known to every surgeon who sees these conditions. In cases of frank perforation of a peptic ulcer, the diagnosis is frequently easy, but in many instances of imminent or forme fruste perforations of a duodenal or gastric ulcer, as well as in cases of trauma to the abdomen with perforation of the small intestine or colon, the percentage of wrong diagnoses is quite appreciable. Any practical means of arriving at an exact diagnosis of whether actual perforation has occurred would be of great value in the treatment of this type of disease.

In 1915, Popper[9] first suggested the x-ray visualization of a subphrenic collection of gas with the patient in an upright position as an aid in the diagnosis of perforated peptic ulcers. In the following year, Lenk[7] noticed the occurrence of pneumoperitoneum following bullet wounds of the abdomen and recommended the use of x-ray examination in the diagnosis of perforations of the gastrointestinal tract. Dandy,[2] in 1919, noted a subphrenic pneumoperitoneum on the chest plate of a patient with a perforated ulcer of the transverse colon. This chance observation later led to the development of ventriculography. Vaughan and Brams[13] in 1925 published the first series of cases of any size in which this method of diagnosis was used. They found that subphrenic accumulations of gas could be demonstrated in 86.2 per cent of 29 proved cases of acute perforation of peptic ulcers. Other series have been reported as follows:

Vaughan and Singer (1929):[14] 63 proved cases of perforated peptic ulcer; 54 cases showed presence of pneumoperitoneum on x-ray examination—87 per cent.

Finsterbusch and Gross (1932):[3] 110 proved cases of perforated peptic ulcer collected from the literature; 95 cases showed presence of gas by x-ray examination—86 per cent.

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Spinzig (1933):[11] 21 proved cases of perforation of the gastrointestinal tract; 21 cases showed the presence of gas by x-ray examination—100 per cent.

Johnson (1937):[6] 42 proved cases of perforated gastric and duodenal ulcers; 35 cases showed the presence of gas by x-ray examination—83 per cent.

Petrén (1937):[8] 40 proved cases of perforated gastric, duodenal, and gastrojejunal ulcers; 27 showed the presence of gas by x-ray examination—68 per cent.

CRITERIA OF PERFORATION

The criteria by which writers have considered free perforations to be proved have been, in practically all cases, the demonstration at operation or autopsy of a perforation in free communication with the general peritoneal cavity, or the demonstration of a pneumoperitoneum by x-ray. A moment's consideration will show that figures drawn from a series of cases based on the above criteria must be somewhat weighted in favor of the accuracy of x-ray diagnosis, since no account is taken of those cases of perforation, such as the sealed or forme fruste type which shows no gas by x-ray examination, which is not operated upon, and which is not autopsied. It should be borne in mind further that failure to find a perforation at operation or during the course of a routine autopsy does not absolutely exclude the possibility of its presence. On the other hand, there are certain objections to the acceptance of the presence of a pneumoperitoneum without confirmation at autopsy or operation as proof of a gastrointestinal perforation in the usual sense. The fact that a ruptured appendix might theoretically give rise to a pneumoperitoneum must not be overlooked. While in our experience such a condition has not been detected in a large number of roentgenograms taken for the purpose of determining whether pneumoperitoneum was present in frank cases of perforation of the appendix, isolated case reports do appear in the literature. Cases of so-called "gas peritonitis," in which a peritonitis with the concomitant formation of free gas in the peritoneal cavity is present, have appeared rather frequently in the recent German literature. One such case has been seen at the University of Minnesota Hospitals.

Case Report.—A forty-three-year-old Mexican woman presented an acute inflammatory lesion in the abdomen. A large unencapsulated pneumoperitoneum was demonstrated by x-ray examination. Under a diagnosis of perforated peptic ulcer, this patient was operated upon. Careful exploration of the abdomen revealed no sign of a perforation of the gastrointestinal tract nor any source of the generalized peritonitis which was present. The patient died, but autopsy was not permitted. In the absence of a carefully performed autopsy, it must be admitted the possibility of a gastrointestinal perforation was not completely excluded.

Rare cases are seen in which gas is found in the peritoneal cavity without symptoms and without obvious cause. One such instance has been observed at this hospital (Fig. 1).

Case Report.—A thirty-one-year-old white woman had complained of vague pain in the lower part of her back for three months. During this time she had several

short attacks of epigastric discomfort with pain radiating to the right shoulder. These attacks usually followed meals and lasted ten to fifteen minutes. At no time had she been confined to her bed. Physical examination was essentially negative except for the absence of liver dullness on percussion of the abdomen. Various x-ray films of the abdomen (Fig. 1) showed the presence of a massive pneumoperitoneum. Roentgen examination of the stomach and colon, as well as a cholecystogram, were all negative. Despite the fact that the patient was afebrile and had no acute complaints or evidences of peritonitis, an exploratory laparotomy was advised. This the patient refused and left the hospital nineteen days after admission. A definite cause for the pneumoperitoneum was never determined.

EXPERIMENTAL OBSERVATIONS

In an effort to obtain data concerning how much reliance might be safely placed on negative x-ray findings in suspected cases of gastric and intestinal perforations, certain observations were made on thirteen patients incidental to the production of a diagnostic pneumoperitoneum.

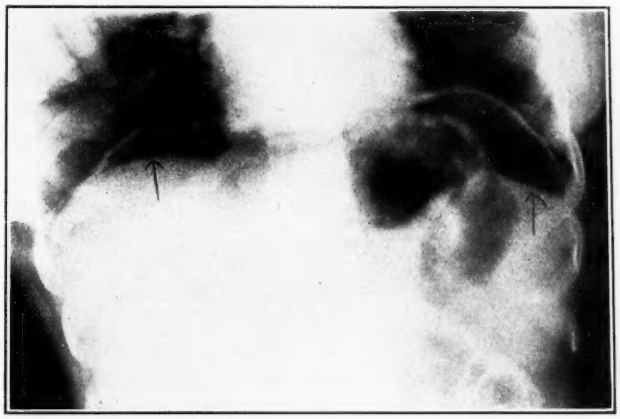

Fig. 1.—Spontaneous pneumoperitoneum, cause undetermined. Roentgenogram of abdomen, upright position, showing gas accumulated (arrows) under each diaphragm. The shadow of the gas-filled stomach and colon and the density of the spleen can be seen on the left side. This appearance is characteristic of pneumoperitoneum whether spontaneous or artificial. In this case no cause for the pneumoperitoneum could be ascertained and there was no evidence of peritonitis.

Similar observations were made on five cadavers shortly after death. These experimental observations were begun at the suggestion of O. H. Wangensteen and had as their purpose the determination, if possible, of how small a quantity of gas could be seen regularly under the diaphragm and, in particular, under the right dome of the diaphragm.

It seems strange that this important point has not been determined before, but the only references to it in the literature are the statement by Vaughan and Brams in 1925[13] that only a few cubic centimeters of air were required to be seen by the x-ray method, and the statement by Friedman in 1934[4] that no definite determination had been made as to the smallest amount of gas that would cast a shadow, but that it was believed that as little as 50 c.c. might be visualized on an x-ray film.

A. *Observations Incidental to Diagnostic Pneumoperitoneum.*—In thirteen patients with abdominal lesions, pneumoperitoneum was produced for purposes of diagnosis. The gas injected was either air, oxygen, or carbon dioxide. Carbon dioxide was used in the majority of the cases as it was found that quantities of this gas as large as 1,600 c.c. were almost completely absorbed from the peritoneal cavity within a few hours. During this time the desired x-ray films could be made and the discomfort which the injection of gas occasionally produced was diminished to a minimum. However, in the preliminary experimental observations made on these patients and reported here, air was used as the injected medium to obviate the rapid absorption of carbon dioxide.*

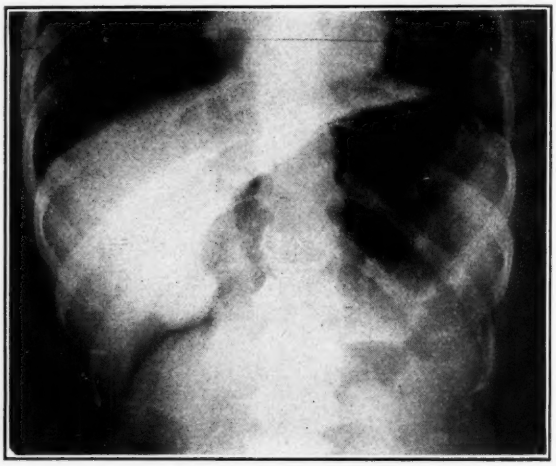

Fig. 2.—Artificial pneumoperitoneum. Roentgenogram of abdomen in upright position demonstrating outline of liver, gallbladder, spleen, and adhesions. Note complete absence of gas under right diaphragm, between left lobe of liver and left diaphragm, and between spleen and left diaphragm. There appears to be a small segment of left diaphragm under which the gas has accumulated. Due to adhesions even the large amount of air injected does not accumulate in the subphrenic spaces.

The abdominal wall, at a point slightly below the umbilicus and about two inches to one side of the midline, was infiltrated with 1 per cent novocaine. The patient was then placed on the fluoroscopic table in the vertical position and small measured quantities of air were injected into the peritoneal cavity by means of a needle inserted through the anesthetized area. During the time of the injection and at frequent intervals immediately afterwards, the site of injection and the subdiaphragmatic areas were observed through the fluoroscope. Similar injections and observations were also made with some of the pa-

*Other observations have shown that 1,200 to 1,600 c.c. of carbon dioxide are completely absorbed from the peritoneal cavity in twenty-four hours. Similar quantities of oxygen are absorbed within four to seven days. The same amount of air, as demonstrated by x-ray examination, will not be completely absorbed for as long as two weeks after injection.

tients in the left lateral decubitus position; i.e., a posteroanterior projection with the patient lying on the left side.

The results of these observations showed that when small quantities of air (10 c.c.) were injected, all, or portions, of it frequently remained localized for a short time at the site of injection. Usually after a few seconds, particularly if the abdomen was massaged or if the patient took several deep breaths, this gas tended to accumulate beneath the diaphragm. In the average case 20 to 30 c.c. of air had

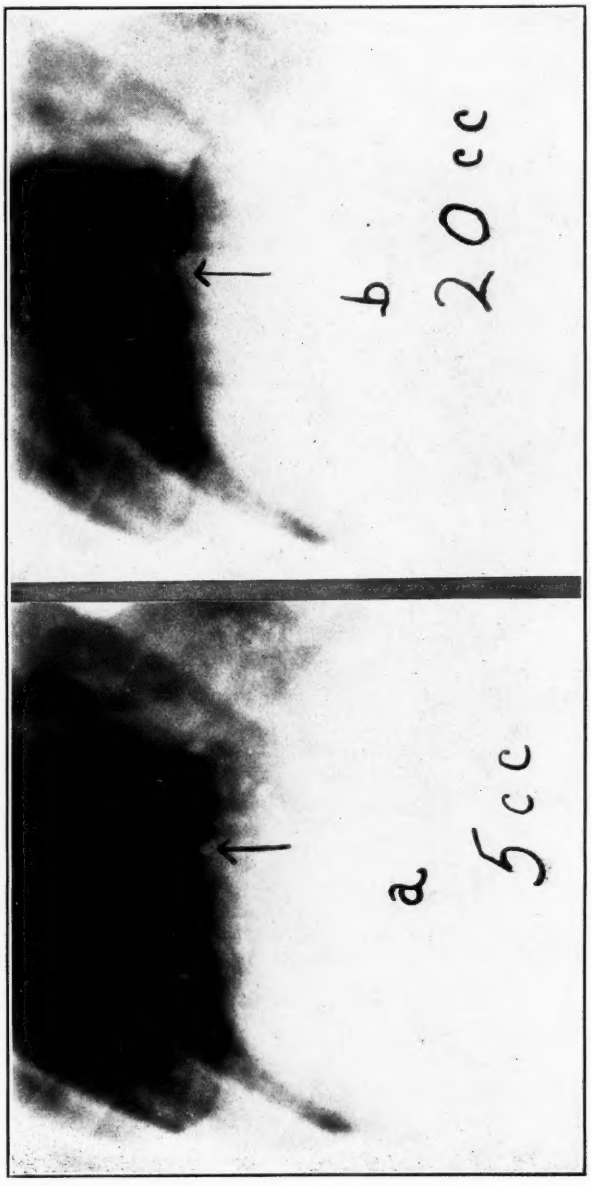

Fig. 3.—*A*, Experiment on cadaver; roentgenogram after introduction of 5 c.c. of air under the right diaphragm showing clearly the presence of gas. *B*, Same experiment as *A*, showing increased visualization of air after injection of 20 c.c.

to be injected before an unmistakable subphrenic accumulation could be seen. In the occasional case quantities of air as large as 50 c.c. had to be used and in these cases the place where it was often seen first was lateral to the right lobe of the liver instead of above it. It was our impression that air tended to accumulate more frequently and in larger amounts under the right dome of the diaphragm than under the left. In any event, a small quantity of air was more frequently and more quickly observed under the right dome than under the left. In one case, despite the establishment of a large diagnostic pneumo-

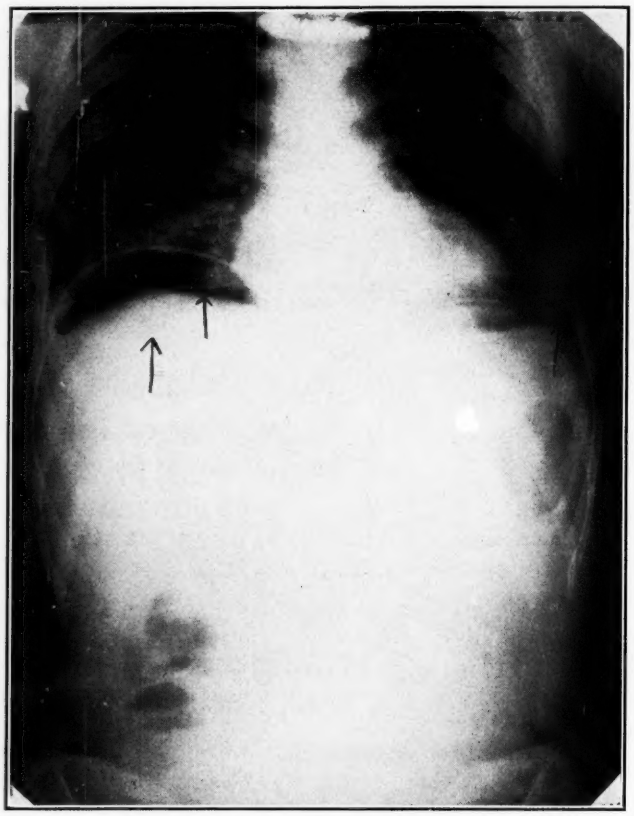

Fig. 4.—Spontaneous perforation of duodenal ulcer. Roentgenogram of abdomen in upright position showing characteristic accumulation of gas under both diaphragms. The presence of fluid in the peritoneal cavity is also shown by the dense horizontal shadow beneath the gas, i. e., the fluid level.

peritoneum of 1,400 c.c. of carbon dioxide, no gas ever appeared under the right diaphragm and only a small portion under the left (Fig. 2). At that time, this observation was interpreted as indicating extensive adhesions between the under surface of the diaphragm and the liver and spleen. A subsequent laparotomy demonstrated the presence of an extensive tuberculous peritonitis. It is obvious that in cases of this type examination in the upright position, as usually done, would fail entirely to reveal gas. Examination in the left lateral decubitus

position is of very great value under such conditions as gas can thus be demonstrated between the lateral abdominal wall and the liver even if the subphrenic space is sealed.

B. *Observations Made on Cadavers.*—The above observations indicated that small amounts of air injected into the peritoneal cavity of the average patient at a point some distance from the diaphragm probably did not reach the subphrenic spaces immediately or in their entirety. The following experiments were therefore performed on five cadavers within two hours after death.

The cadaver was first strapped to the fluoroscopic table and then placed in the vertical position. A small portion of the ninth or tenth

Fig. 5.—Same case as shown in Fig. 4. Posteroanterior roentgenogram in left lateral decubitus position showing shifting of gas which occupies the space between the lateral abdominal wall, the diaphragm, and the liver. The fluid level has also shifted so that it is parallel to the long axis of the body.

rib in the midaxillary line on the right side was resected, and by means of a syringe and hypodermic needle, varying quantities of air were injected through the diaphragm directly into the subphrenic space. As soon as any suggestion of air between the diaphragm and upper surface of the liver could be seen with the fluoroscope, an x-ray film was exposed on the fluoroscopic table.

In two of the cadavers 5 c.c. of air was clearly visualized (Fig. 3*a*), and in all five cadavers 10 c.c. was readily seen without any difficulty (Fig. 3*b*). Similar observations made with the cadaver in the left lateral position indicated that somewhat larger amounts of air (10 to 20 c.c.) had to be injected to be seen regularly.

TECHNIQUE OF X-RAY EXAMINATION IN CLINICAL CASES

Many of the clinical cases to be reported here were examined only in the upright position, some with fluoroscopic observation, others by posteroanterior roentgenograms alone. Whenever possible the films were made at the end of deep inspiration. In the past few years three exposures have been made; one a posteroanterior with the patient upright (Fig. 4), one an anteroposterior with the patient supine, and one a posteroanterior with the patient lying directly on the left side with the right side up (Fig. 5). In the past year these films have been made with the Potter-Bucky diaphragm; prior to that they were made without it.

ANALYSIS OF CLINICAL CASES

At the University of Minnesota Hospitals a diagnosis of perforation of the stomach, duodenum, jejunum, ileum, or colon was made in 70 instances during the ten-year period, 1927 to 1937. Forty-seven of these 70 cases were submitted to x-ray examination to determine the presence or absence of a subphrenic pneumoperitoneum. In 56 of these 70 patients the presence of a perforation was demonstrated at either operation or autopsy. In 6 other cases the actual proof of perforation was dependent upon the demonstration of a pneumoperitoneum by x-ray examination. In the remaining 8 cases the diagnosis of perforation was based on the clinical findings and physical examination alone. These 70 perforations were divided with respect to anatomic location as follows:

ANATOMIC LOCATION OF PERFORATIONS OF GASTROINTESTINAL TRACT: UNIVERSITY OF MINNESOTA HOSPITALS, 1927-1937

		DIAGNOSIS CONFIRMED BY:		
		OPERATION OR AUTOPSY	X-RAY EXAMINATION ALONE	HISTORY AND PHYSICAL EXAMINATION ALONE
Stomach	14	11	1	2
Duodenum	43	33	4	6
Small intestine	4	4	–	–
Colon	9	8	1	–
		56	6	
Total	70	62		8

A. *Perforations of the Stomach.*—In the 14 instances of perforation of the stomach, 12 peptic ulcers, and 2 carcinomas, definite proof of perforation was obtained in 12. Only 8 of these cases, however, were submitted to x-ray examination and in 6 cases only was free gas demonstrated in the peritoneal cavity. In other words, the x-ray diagnosis of perforation was only 75 per cent correct in this small series of cases. One of the 2 proved cases of perforation in which x-ray examination failed to demonstrate a free pneumoperitoneum was a 4 mm. perforation on the anterior surface of the pyloric portion of the stomach

which had occurred five hours before the x-ray examination was made. The x-ray films taken in the other case were poor exposures, but we are unable to say whether failure to demonstrate a pneumoperitoneum was dependent on this poor technique or some other factor. With small quantities of gas, films of poor quality may readily prevent a correct diagnosis.

In the 6 cases in which a pneumoperitoneum was demonstrated by x-ray study, gas was seen under both sides of the diaphragm in every instance (Fig. 6). Roentgen examination was made with the patient in the left lateral decubitus position in only two instances. (Fig. 7.)

B. *Perforations of the Duodenum.*—Thirty cases out of a total of 37 proved cases of perforation of the duodenum at the site of duodenal ulcers were submitted to x-ray examination. Free gas in the peritoneal cavity could be demonstrated in only 22 instances, or 73.3 per cent. An x-ray film with the patient in the vertical position was made in each of the 22 cases (Fig. 5). A film with the patient in the left lateral decubitus position as well as the vertical position was made in only 14 instances (Fig. 6). In 3 cases free gas could be seen on the upright plate, but not on the decubitus film. In 1 case free gas could be seen on the decubitus film, but not on the upright. Of the 21 cases in which a free subphrenic pneumoperitoneum was present in the upright film, it was present under the domes of both diaphragms in 10 instances; under the right dome but not the left, in 9 instances; and under the left dome but not the right, in 2 instances (Fig. 8).

One of the 8 proved cases of perforation of the duodenum which did not show the presence of free gas in the peritoneal cavity by x-ray examination was an extremely obese moribund patient in which the x-ray films were not clear enough to be read satisfactorily. Failures in diagnosis due to such a cause have been included in our series, since for practical purposes they constitute a valid objection to the use of the x-ray method of diagnosis.

No adequate explanation of the failure to visualize gas by x-ray examination in the other 7 cases of duodenal perforation is apparent. All were perforations of duodenal ulcers in the anterior wall of the first or second portions of the duodenum. The average size of the perforation was 5 mm. The average time interval between perforation and x-ray examination was five and one-half hours.

C. *Perforations of the Small Intestine.*—A diagnosis of perforation of the jejunum or ileum was made in 4 instances. Each of these perforations was demonstrated at either operation or autopsy examination. Two of these 4 cases were submitted to x-ray examination. One was a perforation of a tuberculous ulcer in the distal portion of the ileum. This case was seen at the hospital and x-ray examination made sixteen hours after the perforation had probably occurred. Free

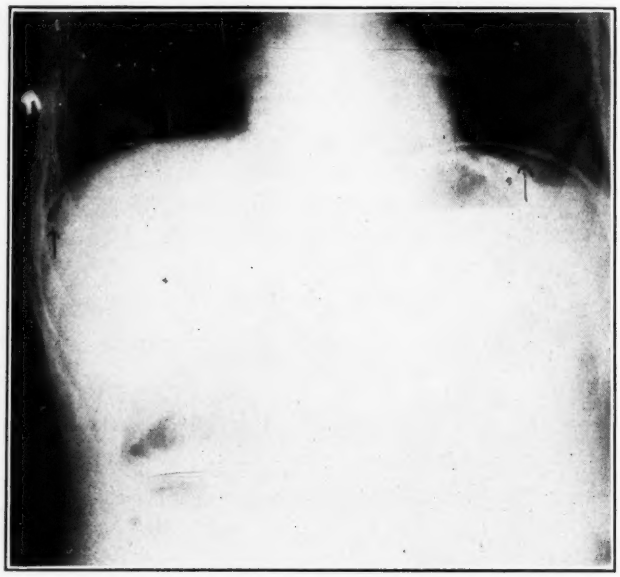

Fig. 6.—Spontaneous perforation of gastric ulcer. Roentgenogram of abdomen in upright position showing moderate accumulation of gas under the left diaphragm (arrow). The gas and fluid in the cardia of the stomach are shown below the shadow of the gas. On the right there is a much smaller amount of gas (arrow) which does not spread through the whole subphrenic space.

Fig. 7.—Same case as shown in Fig. 6. Posteroanterior roentgenogram in left lateral decubitus position showing shifting of gas under right diaphragm to a more caudad position (arrow). It should be noted that the shadow of the gas on the right side is more obvious in this film than in the one made with the patient upright.

gas was seen under both domes of the diaphragm in a roentgenogram taken with the patient in the upright position. The other case was that of a boy with a perforation of the jejunum. The first examination, eight hours after the perforation, failed to show any signs of a pneumoperitoneum. A second roentgen examination, sixteen hours later, showed the presence of gas beneath the left dome of the diaphragm.

The results of the x-ray examination in these 2 cases agree well with our clinical impression that in early perforations of the small intestine roentgenography is of much less value in diagnosis than in those cases where a considerable interval has elapsed between the perforation and the x-ray examination.

Fig. 8.—Perforation of duodenal ulcer. Roentgenogram of abdomen in upright position showing a large amount of gas under the left diaphragm, while none can be seen on the right side. The confusing shadows on the left side caused by the gas in the stomach and colon are apparent.

D. *Perforations of the Colon.*—The experience of any large hospital attests the fact that the most common cause of perforation of the colon is obstruction of that organ. This is brought about by the fact that in the great majority of patients the ileocecal sphincter and valve effectually precludes the retrograde passage of feces and gas which gain entrance to the colon or are produced there. An increasing intracolonic pressure, such as occurs in obstruction, will in time produce necrosis and rupture of the cecum by stopping the flow of blood through the vessels in its walls. Thus the mechanism and clinical course of perforations of the colon are different from the usual mechanism and clinical course of perforations of the stomach and

duodenum. We have included them, however, for the sake of completeness. Nine cases of perforation of the colon have been observed. Eight of these perforations have been verified by operation or postmortem examination. Proof of perforation in the remaining case rests on the visualization of a free pneumoperitoneum on an x-ray film. An x-ray examination was made in 7 of these 9 cases of perforation and free pneumoperitoneum was seen in all of them. The cause of perforation was carcinoma of the colon with obstruction in 5 cases; obstruction of the sigmoid flexure in a strangulated left indirect inguinal hernia, in 1 case; rupture of the uterus which was adherent to the sigmoid flexure, in 1 case; perforation of an ulcer of the colon, the etiology of which was not determined, in 1 case; and the accidental perforation of the wall of the colon with a suture in the closure of a laparotomy incision, in 1 case.

None of the 7 cases of perforation of the colon which were submitted to x-ray examination were early perforations. The time interval between perforation and x-ray examination was over twenty-four hours in every case and as long as several days in some cases. Four of these 7 perforations occurred in the presence of obstruction of the colon. These two factors, i.e., a lapse of at least twenty-four hours between perforation and x-ray examination and the presence of obstruction with the concomitant large accumulation of gas in the colon at the time of perforation, may explain the fact that roentgenography was 100 per cent correct in the diagnosis of these lesions. It is quite doubtful that an equal degree of accuracy is attained by roentgen examination in early cases of colonic perforation or in cases occurring in the absence of colonic obstruction.

FACTORS TENDING TO DECREASE THE ACCURACY OF X-RAY DIAGNOSIS IN PERFORATIONS OF THE GASTROINTESTINAL TRACT

One may well ask that if such small quantities of gas as 5 to 10 c.c. can be consistently seen in the subphrenic space on an x-ray film, why is not the accuracy of diagnosis by x-ray examination in cases of intra-abdominal perforation of the gastrointestinal tract even greater than the usually reported 85 per cent. The answer to this question cannot be stated with certainty, but several factors seem to merit consideration.

If one assumes that a very small quantity of gas has escaped from the stomach or intestine, it will reach the subphrenic space in a short time, provided the space has not been obliterated by adhesions and provided the patient assumes at least a semierect position for two or three minutes before the x-ray examination is made. Rubin[10] has stated that, in the living patient, when an artificial pneumoperitoneum has been created gas collects under the right diaphragm in two or three minutes. Geier[5] has reported a case of perforation of a gastric ulcer in which gas was seen under the diaphragm ten minutes following the perforation.

With very small quantities of gas, however, after it has reached the subphrenic space, the shadow may be obscured by the dome of the liver on the right side or a distended stomach on the left unless the x-rays are directed in a fairly exact vertical direction with respect to the longitudinal axis of the body and unless the longitudinal axis of the body is vertical with the floor at the time the x-ray examination is made. This is purely a question of gravity. Technical difficulties due to unusually large patients, inability of the patient to cooperate in stopping respiratory movements, technical errors which are prone to occur in cases which are examined under emergency conditions, all tend to militate against clear visualization of the faint shadow produced by small quantities of gas.

Those factors, however, which operate to prevent the escape of gas through a perforation are probably of more significance in decreasing the accuracy of x-ray diagnosis. The influence which the concomitant presence of both fluid and gas in a viscus at the time of perforation plays in the escape of gas from that viscus would seem quite appreciable if the site of perforation should happen to be located below the fluid level. Unless the patient assumes a supine or left lateral position following a perforation of the stomach or duodenum, a considerable quantity of fluid may escape before any gas can gain access to the free peritoneal cavity. Occasionally food particles or plugs of mucus become caught in the perforation and serve as an adequate seal until the reaction of the surrounding peritoneum and omentum has effectively localized or walled off the perforation. In some instances the redundant mucosa of the stomach and small intestine may prolapse through the perforation and prevent the escape of gas for a variable period of time. Recent experimental work by Bergh, Bowers, and Wangensteen[1] on the factors influencing the development of peritonitis in perforations of the gastrointestinal tract of dogs has a direct bearing on this question.

THE VALUE OF ROENTGEN EXAMINATION IN THE LEFT LATERAL DECUBITUS POSITION

X-ray examinations of 14 patients with perforation of the duodenum were made with the patient in both the upright and left lateral decubitus positions (Figs. 5 and 7). In no other type of perforation have enough examinations in the left lateral decubitus position been made to furnish a basis for appraisal of this procedure from a diagnostic standpoint. In 3 patients a pneumoperitoneum was seen in the upright position, but not in the lateral position, and in only 1 patient did the examination in the lateral position permit a diagnosis when the upright failed.

The left lateral decubitus position, however, has several advantages. In the occasional case in which the subphrenic space is obliterated by

adhesions, a collection of gas may be seen which otherwise would be missed. The chief criticism of the x-ray method of diagnosing gastrointestinal perforations has been that it was too strenuous a procedure for critically ill patients. While this criticism may or may not have merit as regards this examination in the upright position, certainly little criticism can be made of the use of the lateral decubitus exposure. The patient need not be moved or raised from his bed or litter. All that is required is that he lie on his left side.

The added information gained from the film made in this position, when taken together with the upright, is often valuable in deciding

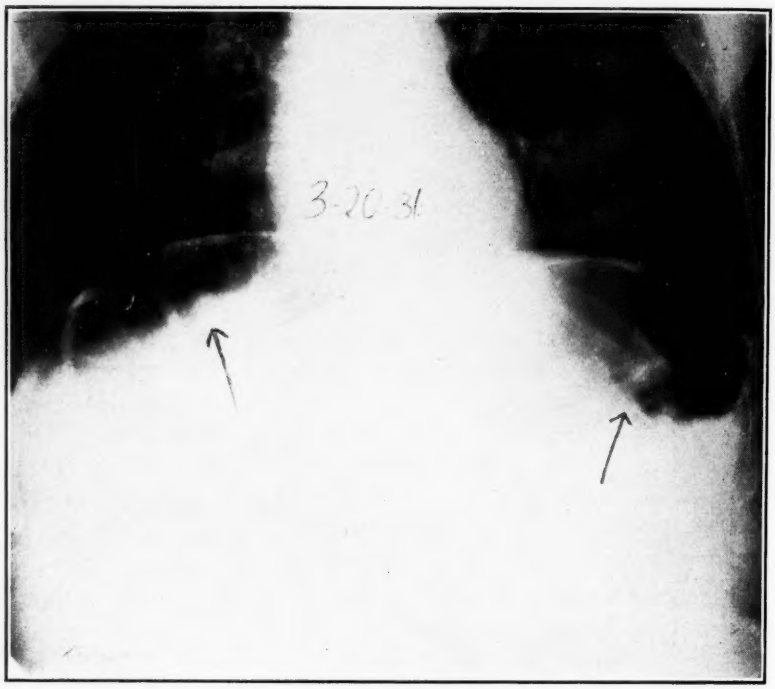

Fig. 9.—Ptosis of the liver and distention of the colon. Roentgenogram of diaphragmatic regions in upright position showing marked accumulations of gas in stomach and splenic flexure of colon (arrow) under left diaphragm. On the right side the liver has been displaced downward by the distended hepatic flexure which simulates the appearance of free gas in the subphrenic space. Note the irregular segmented character of the gas shadow under the right diaphragm which denotes that the gas is within the colon.

whether an accumulation of gas seen under the diaphragm is free in the peritoneal cavity or localized in an abscess (Figs. 4-7). We feel that a gaseous shadow which shifts with change in the patient's position is confirmatory evidence on this point. Collections of gas contained within the stomach or bowel may on occasion cause some doubt as to the diagnosis of a free pneumoperitoneum. This is particularly true in those cases where the pneumoperitoneum is confined to the left subphrenic space. In such cases a stomach distended with gas frequently obscures the pneumoperitoneum and presents a very

confusing picture (Figs. 8 and 9). X-ray films made with the patient in the right lateral decubitus position may be of help in these cases, but they are often of no value because of the multiple gaseous shadows frequently produced in the left upper quadrant by the colon and small intestine. Under such circumstances it has been the practice at the University Hospitals to empty the stomach through a nasal catheter and then reexamine the patient in the upright position. This simple procedure usually suffices to settle this question.

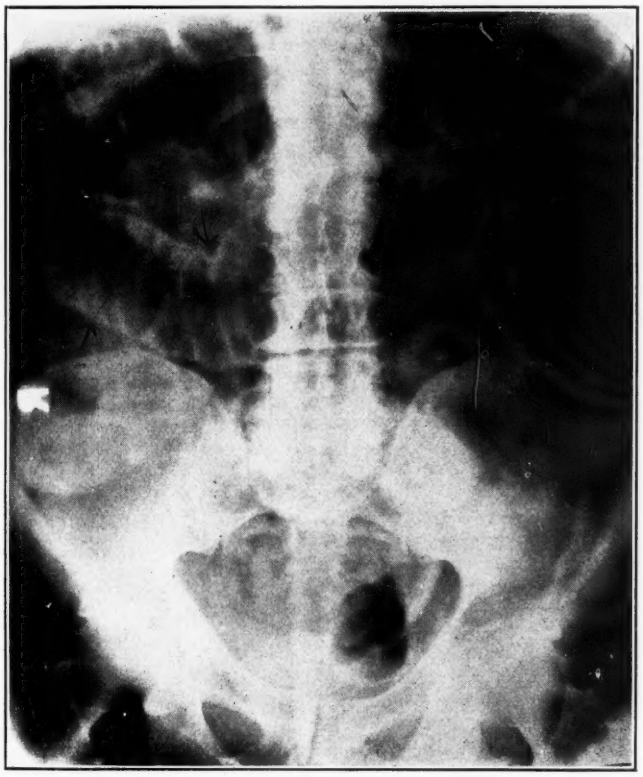

Fig. 10.—Acute obstruction of sigmoid colon from hernia with secondary rupture of cecum and pneumoperitoneum. Roentgenogram of abdomen in supine position showing marked distention of bowel with gas. Note that outer walls of intestine are clearly visible, indicating presence of free gas in peritoneal cavity without the use of upright or lateral decubitus positions.

In rare instances when the liver is ptotic, the hepatic flexure of the colon may be seen occupying the right subphrenic space (Fig. 9). Unless this is kept in mind and the characteristic signs of gaseous accumulations in the colon observed, an erroneous diagnosis of subphrenic pneumoperitoneum may occasionally be made. The fact that air will often remain in the peritoneal cavity for several weeks after laparotomy must also be borne in mind so that a mistaken diagnosis of perforation may not be made in such cases.

In an occasional case, examination must be restricted to films made with the patient in the supine position. Under these circumstances diagnosis is rendered more difficult, but it is well to bear in mind that moderate quantities of gas in the peritoneal cavity will outline the hollow viscera which are usually distended with gas themselves (Fig. 10). Under such conditions the thickness of the wall of the stomach or bowel may be clearly visible, both the inner surface and the serosal surface being delineated by the gas. This may give the clue to the presence of air previously unsuspected.

INDICATIONS FOR OPERATION

A. *Perforations of the Stomach and Duodenum.*—The practice in this clinic is to operate upon all cases of demonstrated perforation except those in which reliable evidence of encapsulation is present. Even late cases without encapsulation are best treated by efforts directed at closure of the perforation. The question of operation in those cases in which pneumoperitoneum is not demonstrated by x-ray or in which it appears to be encapsulated must be decided on the basis of history and physical findings of each case.

All cases in which pneumoperitoneum can be demonstrated in which the history indicates a recent onset should be considered as open leaks into the general peritoneal cavity. In those instances in which some time has intervened (forty-eight hours or more) and the patient's status (especially the pulse) seems satisfactory despite a large pneumoperitoneum, encapsulation may be considered as having occurred. Such a patient warrants conservative treatment. Wangensteen[15] has described such instances and discussed their management by conservative means. Vaughan and Singer[14] have advocated the nonoperative treatment of instances of so-called sealed perforations in which the clinical picture is that of ulcer perforation, but in which no gas can be visualized below the diaphragm on a properly exposed x-ray film. Similar cases have been described by Wangensteen; and in this clinic, in the instances in which the clinical picture of ulcer perforation was present and in which conservative treatment was carried out (suction applied to an inlying duodenal tube, paraoral fluids, hot packs, and subsequent check-up roentgenograms), no patient has come to grief. Our experience, therefore, has been that this practice has been safe. Yet 9 cases (7 perforations of the duodenum and 2 perforations of the stomach), in which an unsealed perforation was found at operation despite negative x-ray evidence, throw legitimate doubt upon the validity of that conservatism. Vaughan and Singer have also described the conservative management of the forme fruste type of perforation, i.e., the patient with the very small pneumoperitoneum who comes after the lapse of some time. In such instances sealing probably occurs soon after the escape of a small quantity of gas from the stomach

or duodenum. Our experience with such cases wholly substantiates the soundness of the conservatism advocated by Vaughan and Singer in such instances.

B. *Suspected Perforations of Small Intestine and Colon.*—That the evidence of roentgenography in perforation (usually traumatic) of the small intestine or of the colon (usually due to tension perforation consequent upon a carcinomatous block) is even less reliable than in perforation of the stomach or duodenum is the common experience of those who have used this agent widely for the detection of such lesions. Wangensteen[16] has called attention to the shortcomings of the roentgen films for this purpose in suspected traumatic perforation of the intestine by nonperforating types of injury. This impression is opposed to the actual experience as recorded in this paper, but the number of cases here reported is small. Proof of the reliability of x-ray evidence for early demonstration of perforation of the small intestine or colon is still wanting and is demanding of further elaboration. The policy in this clinic is to subject suspected and demonstrated perforations to early operation, at which time the perforation is sought out and closed.

SUMMARY

Any means of making an exact diagnosis as to whether perforation of the stomach, intestine, or colon had occurred would be a boon to surgeons treating these conditions and would lower the incidence of operations now performed for acute abdominal conditions.

The use of the x-ray demonstration of subphrenic pneumoperitoneum as an aid in making such diagnoses has been recommended by various men since 1915. The accuracy of the diagnoses made in this way has varied from 68 to 100 per cent in the various series reported. It is probable that the actual percentage is somewhat lower.

By experiments on 5 cadavers it has been shown that at times as small a quantity of gas as 5 c.c., in the right subphrenic space, can be demonstrated on an x-ray film. Ten cubic centimeters can be seen regularly without difficulty.

There have been 62 proved cases of perforation of the stomach, duodenum, small intestine, and colon treated at the University of Minnesota Hospitals between 1927 and 1937. Forty-seven of these cases were submitted to x-ray examination and gas outside the gastrointestinal tract was observed in 78.8 per cent of them. In 38 cases of perforation of the stomach or duodenum, free gas was observed in 28 (73.7 per cent).

The reasons why gas is not seen in every case probably include the following:

1. Adhesions in the right upper quadrant of the abdomen.
2. Location of the perforation below the fluid level of the fluid in the viscus.

3. Absence of gas in the viscus at the time of perforation.

4. Temporary plugging of the perforation with stomach or intestinal content until the perforation is walled off.

5. Prolapse of redundant mucosa into the perforation blocking the exit of gas.

6. Technical failures in the roentgen examination in cases wherein small amounts of gas are present. These include failure to direct the x-rays horizontal to the long axis of the body, failure to have the patient in the upright position for a few minutes prior to the examination and vertical during the examination, and the ordinary difficulties of roentgen technique.

Roentgen examination in the left lateral decubitus position was found to be of value in establishing the diagnosis of spontaneous pneumoperitoneum. This position is useful when the patient cannot be placed upright. However, if pneumoperitoneum is observed at all on the x-ray film, it will be seen between the right dome of the diaphragm and the liver in 90 per cent of the cases.

Roentgen examination of 2 cases of traumatic perforation of the small intestine showed the presence of free gas in the peritoneal cavity; in one case sixteen hours after perforation, in the other only after twenty-four hours.

CONCLUSIONS

1. As little as 5 c.c. of gas in the right subphrenic space can, at times, be demonstrated by roentgen examination under experimental conditions.

2. In cases of perforation of the stomach and duodenum failure to demonstrate gas by roentgen examination may occur in over 25 per cent of the cases.

3. In cases of perforation of the small intestine or colon, if the time interval between perforation and examination is long (sixteen hours or more), or if obstruction is present, a subphrenic pneumoperitoneum is consistently observed by roentgen examination.

4. It is recommended that cases of suspected as well as demonstrated perforation of the gastrointestinal tract, except those in which there is definite evidence of encapsulation, be submitted to early operation.

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PEPTIC ULCERS FOLLOWING EXPERIMENTALLY PRODUCED OBSTRUCTIVE JAUNDICE

A Consideration of the Factors Concerned in Their Production*

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MANY OBSERVERS have demonstrated the presence of duodenal ulcers following experimentally produced obstructive jaundice (Kapsinow, Berg and Jobling, Loewy, Bollman and Mann), and others have shown that duodenal ulcers develop following various procedures for the exclusion of bile, either by itself or together with the other duodenal secretions from the duodenum (Exalto, Mann and Williamson, Morton, McCann, Neuman, Demoor, and Deloyers, Matthews and Dragstedt, Mann and Bollman,[10] Weiss and Hubster). The factors involved in the production of peptic ulcer following obstructive jaundice are obscure. Except for an occasional report in the earlier literature there are few references to gastric secretion in the condition of obstructive jaundice. The records are only briefly reviewed and consist for the most part of observations on cholelithiasis and other diseases of the biliary tract. Hyperacidity, hypoacidity, and anacidity have been found on examination of icteric patients. The few investigators who have been interested in this subject have reported conflicting data; the majority of them, however, are of the opinion that hyperacidity accompanies jaundice in animals.

The theories advanced as to the etiology of peptic ulcer have been summarized recently by McCann, and since the work of Exalto and of Mann and Williamson, many investigators have corroborated the results of their observations on the occurrence of peptic ulcer following exclusion of the duodenal content from the site of emergence of the gastric content. These investigations emphasize the significance of the acidity of the unneutralized chyme in the production of chronic peptic ulcers.

In normal animals it is known that the ulcer-bearing area of the duodenum is subject to a wide range of pH: "It is the site where the onrushes of acidity produced in the normal fundus are met, diluted, neutralized and buffered for acceptance by the intestines."[8] Normal function of this portion of the duodenum depends on the maintenance of an acid-alkali equilibrium. A shift in this equilibrium may result from any one of many factors, and an unbalance may be temporarily or permanently produced in obstructive jaundice. If the buffering

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capacity of the duodenum is diminished without the occurrence of a similar diminution of acidity in the acid chyme, such conditions would appear to be favorable for the formation of ulcer.

The purpose of this study was to determine whether the factors which are known to be of significance in the experimental production of ulcer are operative in the presence of obstructive jaundice and, if so, if they could play a part in the formation of ulcer. My attention was directed primarily to a study of gastric secretion and motility; secondarily, to a determination of the buffering capacity of the content of the isolated duodenum, in vivo and in vitro, following exclusion of bile by ligation of the common bile duct.

A carefully controlled experimental study of gastric secretion and gastric motility before and after ligation of the common bile duct was made by Still and Carlson. Desiccated bile was first taken up in physiologic saline solution, warmed to body temperature, and injected intravenously in amounts varying between 0.125 and 0.5 c.c. per kilogram of body weight. These investigators found that the immediate effect of this intravenous injection of bile was to lower gastric acidity. In only one dog was there a return to normal values of acidity during the two-hour period of observation. Following these control observations, the common bile duct was ligated. After obstruction to the biliary outflow, the average acidity of the continuous secretion was increased over that in controls and hyperacid values were obtained following test meals. The authors concluded that chronic obstructive jaundice is followed by a decrease in the volume of gastric secretion and by an increase in the acidity of the gastric juice.

Selesnjew made esophageal fistulas and carried out gastrostomy on dogs and studied the gastric secretion before and after ligation of the common bile duct. He noted that, following a forty-eight-hour fast, the stomachs of normal dogs were empty except for insignificant amounts of mucus; the stomachs of jaundiced dogs, however, contained small particles of food with bile-stained mucus. In the presence of biliary obstruction gastric juice was of high acidity. Gastric motility was not affected. By means of various test diets he found the gastric acidity was higher in jaundiced than in normal dogs.

Imes studied the neutralizing capacity of the content of the isolated duodenum of animals in the fasting state and following different test diets. The pH and buffering capacity of the combined secretions in the isolated duodenum remained at a fairly constant level (pH 7.1 to 8.15). He found that 100 c.c. of duodenal content required an average of 60 c.c. of tenth-normal hydrochloric acid, with extremes of 40 to 95 c.c., to reduce acidity to pH 4. Following evulsion of the pancreatic ducts, it required only an average of 36 c.c. to reduce 100 c.c. of duodenal content to a pH of 4.

The recent work of Stevens on the buffering capacity of the content of the isolated duodenum in vivo should also be mentioned. He noted that the content of the duodenum of a fasting animal, after isolation, was capable of neutralizing from 1.5 to 1.75 c.c. of 0.5 per cent hydrochloric acid per minute over a period of two hours.

THE EFFECT OF OBSTRUCTIVE JAUNDICE ON THE SECRETORY FUNCTION OF THE STOMACH

Six healthy, adult, trained dogs were used for this study. Under ether anesthesia and with aseptic technique, gastric and duodenal fistulas of the Mann-Bollman[9] type were made. Following a rest period of about three weeks, routine studies of gastric acidity were made after the technique described by McCann. The subcutaneous injection of 1 mg. of

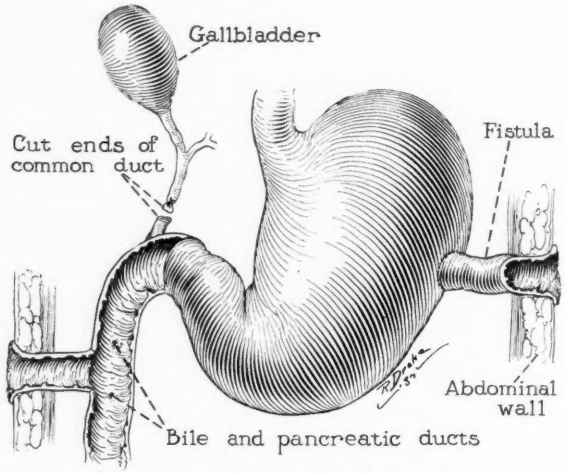

Fig. 1.—Stomach and ileal fistula. The common bile duct has been sectioned and doubly ligated.

histamine and the oral administration of finely diced, lean horse meat were used as stimulants to gastric secretion.

Specimens were collected simultaneously from both fistulas at intervals of fifteen minutes until the gastric acidity and pH had returned to a fasting level. This was accomplished by introducing a soft rubber catheter through each fistula into the stomach and duodenum, respectively. The exact position of the tube in the stomach was determined by roentgenoscopic examination. At each aspiration the entire gastric content was removed and the total volume was estimated. Titration was carried out within a few minutes after each aspiration. The pH was determined by a Leeds-Northrup potentiometer, using the quinhydrone gold electrode. The free and total acidity were determined by titrating clear gastric juice with a solution of tenth-normal sodium hydroxide, using 1 per cent phenolphthalein and 0.5 per cent dimethylamino-azobenzene (Töpfer's reagent) as indicators.

The second operation consisted of doubly ligating and sectioning the common bile duct (Fig. 1). Cholecystectomy was also performed at the same time on four of the six animals. An icteric tint to the sclera was usually noted within seventy-eight hours. Estimations of plasma bilirubin and hematocrit readings were checked every eight to ten days. An icteric sclera and skin, clay-colored stools, bilirubinemia, and biliuria were considered as evidence of occlusion of the common bile duct. Ulcers developed in two of the six jaundiced animals, on the twenty-first and fifteenth days, respectively. One animal survived 38 days and the other, 75 days. Ulcers failed to develop in four dogs, which lived 102, 102, 45, and 32 days, respectively.

The results of a series of observations on these animals may be summarized in a few brief statements: There was no appreciable alteration in gastric secretion following ligation of the common bile duct. The acidity and volume of gastric juice secreted, as well as the duration of secretion following the injection of histamine or a test meal of meat, were similar when tested before and at intervals after operation. The animals in which ulcers did develop and those in which they did not were also similar in this respect.

Although the animals reacted similarly to gastric stimulants, it was observed that the small amount of gastric content that could be aspirated from the normal fasting dog was usually neutral in reaction; whereas, jaundiced animals frequently yielded more fluid which was usually strongly acid in reaction.

THE EFFECT OF BILIARY OBSTRUCTION ON THE BUFFERING CAPACITY OF THE DUODENUM AND ITS CONTENT

In this experiment three healthy dogs were studied. The duodenum was isolated by a two-stage operation, as described by Stevens. Operations were performed under ether anesthesia with an aseptic technique. The first stage consisted of placing an ileal fistula of the Mann-Bollman type in the first portion of the duodenum, and another fistula at, or just distal to, the duodenojejunal junction. In the second stage, the pylorus was sectioned and the duodenal end was inverted. Gastrojejunostomy was performed, with an end-to-side anastomosis approximately 30 cm. from the caudal fistula in the duodenum (Fig. 2). This permitted direct emptying of the stomach into the jejunum, eliminating reflux of the gastric content. The duodenal content remained with its alkaline secretions undiluted.

Specimens were obtained from the proximal and distal fistulas of the animal, both when fasting and after various diets, and the ranges of pH were estimated. Following this, 0.5 per cent hydrochloric acid was introduced into the proximal fistula through a small rubber catheter at a fixed rate by the gravity drip method. Specimens were collected

from the distal fistula at intervals of fifteen minutes and the pH values were noted. Quantitative determinations of chloride ion were made following introduction of the hydrochloric acid when the animal was fasting and after the various diets.

This procedure was repeated on separate days until the maximal amount of acid that could be introduced into the proximal end of the duodenum without materially changing the reaction of the content of the distal portion was determined. This quantity was considered to be the neutralizing capacity of the normal duodenum and its contents. When

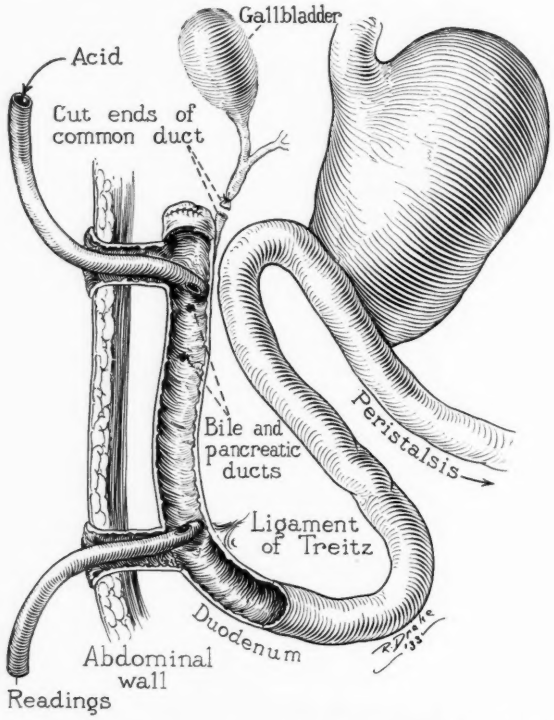

Fig. 2.—A gastrojejunostomy with formation of ileal fistulas; one in the proximal and the other in the distal portion of the duodenum. The common bile duct has been sectioned and doubly ligated.

sufficient data concerning the neutralizing capacity of the duodenum had been obtained, biliary obstruction was produced in each animal by double ligation and section of the common bile duct and the neutralizing capacity of the duodenum was again determined.

Studies of these dogs were resumed on the third to the fifth day after operation. Gastrojejunal ulcers developed in two of the dogs, twenty-eight and thirty-eight days after operation.

The range of pH, as studied from the proximal and distal fistulas of the duodenum in the fasting animals, was approximately the same before and after the exclusion of bile.

Interesting results were noted in the buffering capacity of the duodenal content in vivo and in vitro following the exclusion of bile. In every instance there was a noticeable decrease in the capacity of the duodenum to neutralize acid in the presence of jaundice. The extent of the reduced capacity to neutralize acid after obstruction of the biliary outflow was equivalent to approximately 0.25 c.c. of 0.5 per cent hydrochloric acid per minute for the period of observation.

TABLE I

BUFFERING CAPACITY OF THE DUODENUM AND ITS CONTENT

	IN VIVO*		IN VITRO	
	C.C. OF 0.5% HCl PER MINUTE		C.C. OF N/10 HCl TO REDUCE pH OF 100 C.C. OF ISOLATED DUODENAL SECRETION TO pH 3.5	
DOG	FASTING	AFTER MEAT MEAL	FASTING	AFTER MEAT MEAL
1	1.25-1.50	1.75	75	85
2	1.00-1.25	1.50	72	88
3	1.50	1.75	77	86
	Following Ligation of Common Bile Duct			
1	1.00		105	
2	0.75-1.00		110	88
3	1.25		92	
	Following Development of Ulcer			
1	0.50	0.50-0.75	80	
2	0.75		70	87

*The maximal amount of 0.5 per cent hydrochloric acid per minute that may be infused into the duodenum via the proximal fistula without reducing the pH of the fluid from the distal fistula below 7.0.

The neutralizing capacity of the content of the isolated duodenum, in vitro, following obstructive jaundice was increased (Table 1). This was probably because of the fact that the material obtained from the proximal fistula was undiluted with bile and consisted of succus entericus and, for the most part, pancreatic juice.

In the two animals in which gastrojejunal ulcers of the perforating type were present, there were some interesting changes in buffer values. These changes were associated with the development of ulcer and persisted throughout the experiment. In these dogs there was an additional decrease in the buffering capacity of the duodenal juices (succus entericus and pancreatic juice). This amounted approximately to 0.25 to 0.5 c.c. of 0.5 per cent hydrochloric acid per minute for the period during which observations were made. In the absence of ulcer, 105 to 88 c.c. of tenth-normal hydrochloric acid had been necessary to reduce the pH of 100 c.c. of duodenal content to 3.5 to 4. In the presence of a gastrojejunal ulcer, only 80 to 70 c.c. was required to bring about the same alteration of pH. This close parallelism of the figures obtained in vitro and in vivo deserves mention.

Determinations were made of the values for chloride in the content of the distal fistula in the fasting state before and following the development of jaundice. The average fasting value for chloride ion was 1.7 mg. per cubic centimeter of duodenal content. After the exclusion of bile, the values were increased to an average of 2.66 mg. per cubic centimeter.

Analyses of gastric content before and after the development of jaundice revealed little if any change. In both animals the gastric acidity was diminished following the development of gastrojejunal ulcer.

THE EFFECT OF OBSTRUCTIVE JAUNDICE ON THE EMPTYING TIME OF THE STOMACH

Seven healthy dogs were chosen for the study of the emptying time of the stomach. The general procedure as given below was carried out for a number of weeks for the purpose of training the animals. A test meal of barium was given and the dogs were taken to the fluoroscopic room to accustom them to the darkness and to noises from the roentgen-ray apparatus. By this preliminary preparation they were soon made familiar with a daily routine.

The opaque meal consisted of equal parts of barium sulphate, acacia (10 per cent), and skimmed milk. This barium mixture was administered by mouth without the use of the stomach tube. Examinations were repeated at intervals of four to six days until a fairly constant reading was established for each dog. Roentgenographic examinations were accomplished for the most part without restraint and dogs which had to be gently restrained required the same attention throughout. The stomach was examined within a few minutes following the meal, then hourly for the next two hours, with subsequent observations at intervals of fifteen minutes until the stomach was empty. The average emptying time of the stomach was then determined by repeated observations.

Under ether anesthesia and using an aseptic technique, cholecystectomy, with ligation of the common bile duct, was performed on five of the seven dogs. From the two other dogs the gallbladder was removed and check readings were made to determine any influence on the emptying rate from cholecystectomy alone. At a later date the common bile duct was also ligated in these two animals and further studies were completed.

In the case of the aforementioned five dogs, readings were resumed on the eighth day following operation, with intervals of rest varying from three to six days or more thereafter. Studies were continued as long as the nutritional state and general health of the animals were maintained. The animals were killed for complete examination in 58, 65, 67, 91, and 124 days, respectively. In three animals chronic duodenal ulcers were found.

Following ligation of the common bile duct, the emptying rate was diminished in three dogs, prolonged in one, and in one no appreciable change was noted. In the three dogs in which emptying was more rapid, an ulcer developed in one.

In the animal in which the average time of emptying of the stomach was prolonged, there were two large duodenal ulcers. It is interesting to note that the change in emptying time was only temporary; the time was eight hours during the second week, three hours during the third week, three hours and one-quarter during the fourth week, and three hours and one-half during the fifth week. Thus, excluding the first emptying rate of eight hours, there was no appreciable change following ligation of the common bile duct.

In the two dogs on which simple cholecystectomy was performed there was no change in the emptying rate of the stomach.

SUMMARY

Peptic ulcers occur in a high percentage of dogs in which jaundice is produced experimentally. These ulcers are usually located in the duodenum or appear in the jejunum in animals in which gastrojejunostomy has also been performed. Of the factors which appear to bear some relationship to the formation of the ulcer, the following, which occur after complete biliary occlusion, should be mentioned.

1. The gastric acidity following test meals does not seem to be significantly altered in the jaundiced dog.

2. The acidity and amount of gastric secretion in the fasting stomach is usually found to be increased in the jaundiced dog.

3. The alkalinity of the duodenal content is not materially altered by biliary obstruction.

4. The buffering capacity of the duodenum and its content is definitely reduced after ligation of the common bile duct, and a further diminution occurs when a peptic ulcer is present.

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DIFFUSE, CAVERNOUS HEMANGIOMA OF THE RECTUM

Report of a Case and Record of Necropsy

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THE first known account of hemangiomatous involvement of the rectum was contributed by A. E. J. Barker in 1883. The patient was a man, forty-five years of age, who, when he first consulted Barker in May of 1882, complained of having had periods of rectal bleeding since boyhood. Specular examination of the rectum revealed large folds of mucosa without narrowing of the bowel. There were several shallow, bleeding ulcers, the bases of which presented a mottled, purplish color. The author made a diagnosis of nevoid lipoma of the rectum. The patient failed steadily because of continued loss of blood. At necropsy the wall of the rectum was found much thickened by a "naevoid" growth which gave a purple color to the mucous membrane. The excellent woodcut in Barker's presentation is reproduced here for comparison with our own specimen (Fig. 1).

In Kausch's case, reported in 1916, treatment by posterior excision of the hemangiomatous mass, leaving a permanent stoma, was successful. Examination of the specimen revealed an extensive, cavernous hemangioma of the rectum.

In 1923, Hennig and Schütt reported a case of cavernous lymphangioma of the knee and cavernous hemangioma of the rectum. Necropsy revealed a rectal mucous membrane dark purple in color, inflamed and swollen. The elevation of the "wall" (mucosa?) was caused by thick, spongelike tissue involving the entire thickness of the wall. The microscopic findings conformed to the picture of a typical hemangioma.

In 1923, R. Bensaude and Antoine reported two cases of diffuse, cavernous hemangioma of the rectum. The first was that of a woman, twenty-one years of age, who had had repeated rectal hemorrhages beginning a month and a half after birth. At the age of ten years she was operated on for "hemorrhoids." At the age of fourteen years she underwent first an anal dilatation for fissure, and then an exploratory laparotomy which revealed a large hemangioma of the intestine, passing behind the uterus and disappearing beneath the peritoneum. It had the appearance of a "large bunch of grapes." Nothing curative was done at this time. Pain increased and the patient rapidly became a morphine addict. In 1920, she consulted Antoine, who found her much retarded

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physically and suffering from anemia and amenorrhea. There were frequent rectal hemorrhages. The abdomen was enlarged and tense; there were visible intestinal contractions. Proctoscopic examination for a distance of 30 cm. revealed a marked enlargement of the "anal veins" (external hemorrhoidal veins?). The rectal lumen was obliterated. The mucosa was pale and edematous, but underlying bluish varicosities were seen. When the mucous membrane was swabbed, the cotton applicators became stained with blood, but no bleeding points or ulceration could be

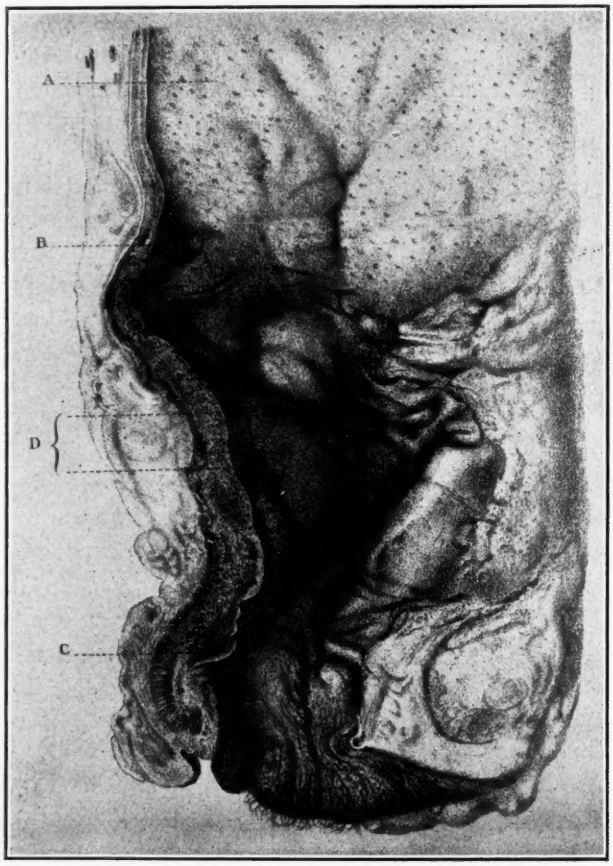

Fig. 1.—This specimen shows about nine inches of the rectum with the anus, preserved in spirits. *A*, normal surface of bowel; *B* to *C*, extent of cavernous nevoid change; *D*, one-half of two ulcers from which fatal bleeding took place. From *B* to *C* the typical cavernous tissue is seen in section. (From Barker, A. E. J.: Cavernous Naevus to the Rectum Proving Fatal in an Adult from Hemorrhage, Med. Chir. Tr., London 66: 229-234, 1883.)

found. Transverse colostomy was performed in January, 1921. The postoperative course was characterized by steady improvement in every respect. The menses were established. At the time of publication of the report the patient was leading a fairly normal life.

The second case of Bensaude and Antoine was that of a man, forty-eight years of age, who entered the hospital May 16, 1921, because of

severe rectal hemorrhages. He had suffered previous attacks of "intestinal" hemorrhages in 1888, 1898, 1904, 1908, and 1915. At the time of the third attack in 1904, the patient was treated for an ulcer of the stomach. In 1908, in the course of the fourth attack, the lower intestine "was suspected"; the suspicion was confirmed by Lockhart-Mummery. Bensaude saw the patient in his last illness and made a diagnosis of diffuse hemangioma of the rectum. The patient died on May 27, 1921, eleven days after admission to the hospital. A report of necropsy was not given.

Bancroft, in 1931, reported a case of hemangioma of the sigmoid with satisfactory relief by colostomy and injection of the superior hemorrhoidal vein (after ligation) with 10 c.c. of 40 per cent sodium salicylate. A second operation, performed eleven months later, comprised: (1) exploratory laparotomy, (2) closure of the colonic stoma, (3) cecostomy, and (4) appendicectomy. The author stated that the condition might have been a true hemangiomatous tumor, or possibly a dilatation of existing vessels. He presented, in addition to his report, an excellent summary of fourteen reported cases and discussed diagnosis and treatment.

R. Bensaude and A. Bensaude, in 1932, reported two cases of rectal hemangioma in which there was also involvement of the perineum, scrotum, and penis. They mentioned a similar case described by Esau, and one described by Kausch; the latter case was that of a female patient with involvement of the labia majora. They designated, therefore, two types of hemangioma of the rectum: (1) the Barker-Kausch type, which is anorectal, or anorectosigmoidal without cutaneous involvement; (2) the Esau-Bensaude type, which is mucocutaneous or genitoperineorectal. Of fifteen cases, eleven were of Type 1 and four were of Type 2.

REPORT OF CASE

A man, aged twenty-eight years, first registered at the Mayo Clinic, Sept. 9, 1925, at the age of sixteen years. His chief complaints were of rectal bleeding and attacks of diarrhea. The trouble began, according to the mother's statement, in the patient's tenth year. The patient was usually constipated, but there were occasional short periods of so-called diarrhea which followed administration of a cathartic. Varying amounts of bright blood were passed at irregular intervals. The patient complained occasionally of pain low in the rectum at the time of defecation.

A year and a half prior to admission to the clinic the patient had an attack of painless hematuria lasting two weeks. He was confined to bed for six weeks with fever and malaise. The significance of this episode will be noted in the report of necropsy.

General physical examination gave essentially negative results. On proctoscopic examination (24 cm.), submucous nodules or lumps seemed to be distributed diffusely and to involve all coats of the bowel. Associated perirectal thickening imparted a sense of fibrosis and limited the mobility of the bowel. The sigmoid was not involved. The disease was believed not to be primary in the rectal mucosa. Biopsy was advised. Diagnosis was withheld.

A specimen was removed for biopsy and the pathologic report was "inflammatory tissue." A course of roentgen therapy was outlined and the first two treatments were given shortly before the patient was dismissed from the clinic.

The patient was registered a second time at the clinic, June 18, 1926. During the period of eight months since his first visit to the clinic he had gained 17 pounds (7.7 kg.) in weight and 2½ inches (6.3 cm.) in height. There was no change in the condition of the rectum and lower sigmoid as observed through the proctoscope.

On Jan. 5, 1937, the patient again visited the clinic. He was pale and weak and suffering from a severe cold. Rectal bleeding had persisted, at intervals, since the time of the previous visit. Hospitalization and supportive treatment were advised.

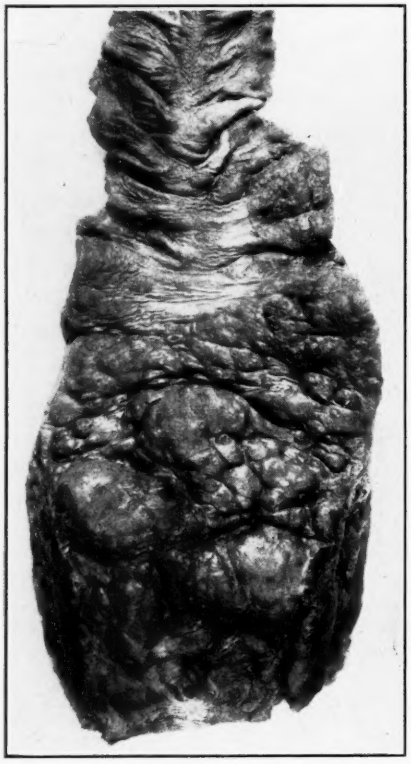

Fig. 2.

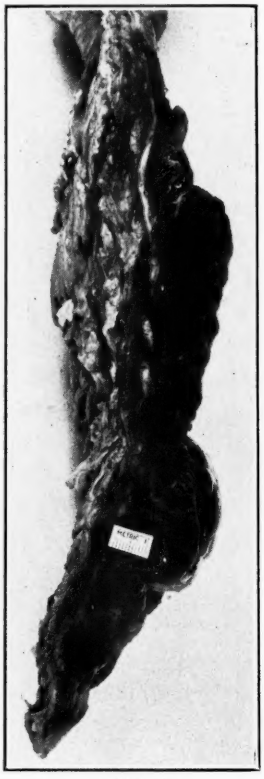

Fig. 3.

Fig. 2.—Anus, rectum, and lower sigmoid laid open. The rectal mucosa is elevated and thrown into large folds by the underlying hemangiomatous masses. The transition to normal bowel is abrupt.

Fig. 3.—The specimen represented in Fig. 2 has been sectioned longitudinally. The mucosal surface is to the right. On the left is shown the perirectal fat. Beneath the fatty layer, and just external to the muscular layers, are shown several large, thrombosed veins. The submucosal layer is markedly exaggerated, as shown by the metric scale, because of its involvement by hemangiomatous tissue. The resemblance to erectile tissue is striking.

When the patient's general condition was sufficiently improved, examination was made under anesthesia and the examiner considered that there was a collection of cystic masses which involved the submucosal tissues of the wall of the bowel. The muscular structures possibly were involved also. The masses varied from growths 6 or 8 mm. in diameter to tumors 2 to 4 cm. long and 1 to 2 cm. wide,

projecting 1 cm. or more above the surface of the wall of the bowel. The masses all felt cystic but could not be entirely obliterated by pressure. The mucosa was adherent to the masses everywhere and there was no place where this membrane appeared to be normal. There was no ulcerous development, except in two places where needle punctures recently had been made. The color, which appeared to come through the mucosa, was bluish gray and gave the impression that the tumors were either hemangiomatous or melanotic. On account of the long duration of the condition, it was unlikely that the tumors were melanotic. The mucosa overlying one of the lowest masses was split. There was profuse bleeding, which, however, was controlled by pressure and by suture. The inner structure of the mass appeared very much like that of the corpus spongiosum. A specimen was taken for microscopic examination. The examiner stated that the advisability of radium

Fig. 4.—Gross section, stained with hematoxylin-eosin. Mucosal surface is to the right. Anal skin appears at the bottom. The muscular coat is shown passing through the center of the section. In the fatty layer (to the left) are seen several large veins, some of them thrombosed. In the submucosa are shown the large spaces, lined by endothelium and filled with blood.

therapy was to be considered and that if such treatment should prove ineffectual, posterior resection and establishment of a permanent stoma might be the only means of saving life. He said, however, that such an operation would be done at very great risk. In the pathologic report it was stated that the tissue was hemangioma. Three transfusions of blood were given. There was only temporary benefit. On Feb. 14, 1937, the patient suddenly died.

At necropsy there was no evidence of hemorrhage. Nothing was found to account for the sudden death. Hemangiomatous involvement of the pelvis and parenchyma of the right kidney explained the painless hematuria.

Only the pathologic changes which pertain to the subject at hand will be mentioned here. Beginning at the dentate margin, there was a series of nodular,

submucous projections extending upward for 30 cm. These projections extended into the lumen for 3 cm. The nodules were irregular in size and constituted about five large masses, measuring from 3 to 5 cm. in diameter, which were gathered under the mucosa like grapes. The remainder of the mucous membrane was raised about 1 cm. from the muscularis. The mucosa at the dentate margin was ulcerated; the largest ulcerated area was about 3.5 cm. in diameter. The remainder of the mucosa was greenish black, a color similar to that of the content of the bowel. In the submucosa and the perirectal fat were many dilated networks of veins, some of which were thrombosed (Figs. 2 and 3). Microscopic examination disclosed large sinuses lined by endothelial cells and filled with blood (Fig. 4).

The anatomic diagnosis was hemangioma of the rectum and of the pelvis of the right kidney.

COMMENT

The tumor in this case, therefore, was a diffuse, blood vascular mass, composed of blood-filled spaces and resembling erectile tissue, which had invaded all the tunics of the rectum except the mucosa—hence the name "diffuse cavernous hemangioma."

Bensaude and Antoine have classified hemangiomas as simple and cavernous. The latter type may be either circumscribed or diffuse. Circumscribed cavernous hemangiomas have little tendency toward invasion. They are not rare in the gastrointestinal tract but involve the inside of the cheeks, the lips, the soft palate, the tongue, the stomach, the duodenum, the small and large intestine, and the rectum. Diffuse cavernous hemangiomas have a tendency toward invasion and a predilection for the rectum and sigmoid. The gravity of this condition is owing to severe and frequent hemorrhages.

Several hypotheses relative to the etiology of this condition have been set forth. The most logical conception, however, and one to which the majority of authors on the subject subscribe, is that the entity is congenital in origin.

The pathologic anatomy in our case coincides closely with the descriptions given by Barker and by Kausch. The tumor is composed of blood vascular tissue characterized grossly by rather large spaces filled with blood. The resemblance to erectile tissue is striking. The sinuses are lined by endothelium. The tendency toward invasion of the wall of the bowel is well shown in our specimen. The overlying mucosa is adherent to the tumor but is intact.

The clinical course is characterized particularly by bleeding from the rectum. In several of the previously reported cases, and in our own, the bleeding appeared in early childhood. The losses of blood vary both in frequency and in amount. The result is severe hypochromic anemia. There may be considerable so-called rectal pain. Our patient complained bitterly of tenesmus and pain in the rectum during the final stages of his illness. In several reported cases the patient has died exsanguinated. The sudden death of our patient has not been explained.

The diagnosis of this condition should be determined by proctosigmoidoscopy and biopsy. Digital examination reveals the nodular characteristic of the involved wall. When the proctosigmoidoscope is first introduced, the lumen of the rectum seems to be almost entirely obliterated, yet the instrument usually can be passed without difficulty and an adherent but intact mucous membrane can be seen. The tumors appear bluish through the mucosa, giving a clue as to their blood vascular structure. Further evidence of the vascular character of these masses can be obtained by aspiration of blood from them. Roentgenologic examination may reveal a filling defect in the rectum. However, roentgenologic study of the rectum is not conclusive and the expert proctoscopist usually can make a more adequate study of this organ. That part of the large bowel beyond the reach of the proctosigmoidoscope can be studied by the roentgenologist, but if a lesion is suspected in the rectum or sigmoid, especially when there have been episodes of obstruction of the bowel, it is much safer to make endoscopic examination before barium has been injected for roentgenologic study.

Treatment by radiation has no value. Surgical treatment, that is, establishment of a permanent colonic stoma, followed if possible by radical excision of the affected part of the bowel, is the only means of relief available at present. Antianemic therapy, transfusions, and other supportive measures are essential in preoperative preparation. In the first case reported by Bensaude and Antoine (1923), colostomy alone sufficed to accomplish marked improvement. The great risk of surgical operation in such cases has been mentioned.

The ultimate prognosis, as well as the immediate surgical risk, is grave. Hemangiomatous involvement of other structures, as illustrated by our own case, may be partially responsible for the poor prognosis. It is probable that had our patient survived radical treatment, ultimately he would have been faced by the necessity of removal of the right kidney because of the hemangioma affecting its pelvis and parenchyma.

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SUBPHRENIC ABSCESS

Factors in the High Mortality

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ABSCESS of the subphrenic region is a rather uncommon lesion. At the Los Angeles General Hospital, during a ten-year period from 1926 to 1936, there were only fifty such infections proved by either surgery or autopsy. During this time the average yearly admissions to the surgical services were over 5,000 patients. Relatively few surgeons encounter a sufficient number of such cases to make practical deductions as to their management. To the man in general practice doing occasional surgery, this complication often brings a feeling of helplessness and dread.

That this feeling is justified may be gathered from reports of mortality in subphrenic abscess, which generally vary from 30 per cent to 80 per cent. The mortality in the fifty cases at the Los Angeles General Hospital, operated and nonoperated, was 50 per cent. With his report of 9.7 per cent mortality, Alton Ochsner has pointed out that more favorable results are possible.

The improvement in the management of these abscesses, as emphasized particularly by Ochsner (an improvement that has led to the lower mortality figures), has not, however, been generally recognized and followed.

For surgical purposes, Ochsner has described the subphrenic space as the area located between the diaphragm above and the transverse mesocolon and transverse colon below. This area is divided by the liver into a suprahepatic and an infrahepatic portion. The suprahepatic space, the more important of the two, is divided into one extraperitoneal space (the bare area) located within the confines of the coronary ligament, and several intraperitoneal spaces. The falciform and round ligaments divide the intraperitoneal spaces into right and left ones. On the left side, the left prolongation of the coronary ligament, the left lateral or triangulate ligament, passes backward to lie at the posterior edge of the liver. This gives rise to only one left superior space. On the right side, the right lateral ligament passes anteriorly, dividing the right superior space into an anterior and posterior.

Of the abscesses in this series, all were on the right side. The majority were in the posterior-superior space. Following appendicitis the

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lesions were almost always in this space; whereas, subphrenic abscesses complicating perforated peptic ulcer were in either the superior or inferior regions.

The etiology in practically all cases was some abdominal infection. The trio, perforated peptic ulcer, appendicitis, and cholecystitis, accounted for over 70 per cent of the abscesses. Perforated ulcers came first in the list, being responsible for 18 of the 50 cases (36 per cent). Six of these developed in individuals who either refused primary surgery or, because of atypical findings, were operated upon for their perforation only after a delay of several days, or not at all.

A fact that seems worth recording is that in all instances of operation for a perforation rubber tissue drains were placed in the upper abdomen. In all but 2, the perforations were merely closed without further surgery. In 1, a Billroth II resection was performed, and in another, a Judd type of pyloroplasty was done.

Thirteen of the 50 cases (26 per cent) followed acute appendicitis or appendectomy and constituted the second largest group. Two of the appendices were not acutely inflamed (one being removed following a subtotal hysterectomy), indicating that the technique of appendectomy might be improved. In the great majority of cases there was perforation of the appendix with a complicating generalized peritonitis. In one instance a subphrenic abscess was not suspected until a year after the removal of a perforated appendix, when the sudden evacuation of foul pus through a bronchus led to further examination.

Although gallbladder disease is not commonly considered as a cause of subphrenic abscess, surgery on the gallbladder or perforation of an acutely inflamed gallbladder gave rise to 6 such infections (12 per cent). Two of these followed cholecystectomy and both had biliary duct drainage. Five of the 6 died. All of these abscesses were accessible to drainage through an anterior approach.

The fourth group (4 cases, 8 per cent) developed from perforations of amebic liver abscesses. All of these gave preceding history of vague abdominal or gastrointestinal distress of many years' duration. In them the subphrenic abscess responded to treatment and all recovered.

Finally, there were 8 cases associated with miscellaneous lesions; 2 resulted from a perforation of a diverticulum of the large bowel; 2, from injury to the liver (automobile accidents); 2, from infected abortions; 1, from a pleural empyema with multiple rib resections; and 1, from a urethral stricture with perinephritic abscess.

The recognition of a subphrenic abscess, as indicated by a study of the cases in this series, is quite difficult, and this difficulty of recognition is apparently one of the chief factors responsible for the high mortality. Approximately one-third of the fatal cases were not operated upon, chiefly because the subphrenic abscess was not recognized.

Some of these cases, however, were complicated and the patients probably would not have recovered even after the benefit of drainage. Since over 60 per cent of the abscesses resulted either from perforation of a peptic ulcer or inflammation of an appendix, merely thinking about the subphrenic infection as a possible postoperative complication would help considerably in its recognition in connection with these catastrophies. The experience of this series indicated that when a subphrenic abscess was suspected it was usually present.

The following cases of subphrenic abscess are of interest, from the standpoint of difficulty of recognition and delay in treatment.

CASE REPORTS

CASE 1.—Male, aged twenty-three years, was operated upon July 22, 1932. A right rectus incision was made and a gangrenous perforated appendix with generalized peritonitis found. Appendectomy with drainage was performed. The patient continued to run a septic temperature postoperatively, otherwise he seemed all right. On Aug. 28, 1932, he developed a severe cough with blood-tinged sputum, dyspnea, and cyanosis, and he was believed to have pulmonary embolism. The patient died Aug. 31, 1932.

Autopsy: Large subphrenic abscess, right posterior walled-off pleural empyema in region of right base, and hepatization of right base. Both abscesses contained gram-negative rods and gram-positive cocci.

CASE 2.—Male, aged thirty-eight years, entered the hospital Oct. 23, 1932, with history and findings typical of perforated peptic ulcer. X-ray examination showed air under the right diaphragm. Laparotomy revealed a great deal of cloudy fluid and small perforation at the pylorus. The perforation was closed with gastrointestinal purse-string sutures; all free fluid was aspirated and a Penrose drain was placed in the subphrenic space. The patient improved, but ten days later he began to run septic temperature. X-ray indicated high right diaphragm, and the white blood count was 16,000. The patient complained of chilly sensations and pain over lower right thorax. A tender mass became palpable below right costal arch. On Nov. 20, 1932, an incision paralleling the costal arch was made and the abscess cavity drained. The patient died a few hours after surgery. Autopsy was not obtained.

In Case 2 it is worth noting that a drain was placed in the upper abdomen and that the abscess was not evacuated until almost a month after the onset of the septic temperature.

Particularly striking about the manner of death of some of these cases, especially those in whom the presence of subphrenic pus was not recognized, is the fact that it was generally sudden and dramatic, suggesting some new accident. The patient appeared fairly well at one moment, but in a short time became dyspneic, cyanotic, and cold. The pulse was rapid and thready. In such cases the diagnosis often made before death was pulmonary embolism.

Delay in instituting surgical drainage of the abscess is quite commonly noted in a study of these histories and is apparently due to the

uncertainty on the part of the surgeon concerning the presence of pus. However, from a study of the case histories, the diagnosis would seem to be quite evident, but in many instances delay was permitted. Usually there is a history of a recent attack of acute appendicitis or perforation of a peptic ulcer. The temperature curve is of the septic type and is associated with leucocytosis. X-ray shows a high diaphragm on one side; a lateral view may show this elevation to be anterior or posterior; and on fluoroscopy the diaphragm is often fixed at the place of elevation. These examinations are frequently repeated in the hope that improvement will follow; drainage is delayed or not done. This delay is the second factor in the high mortality.

Four of our cases of abscesses associated with gallbladder lesions occurred following perforation in acute cholecystitis treated by the method of watchful expectancy.

In the method of draining the abscess is to be found the third, and probably the most important, factor, in the high mortality of the present group of cases. Drainage was accomplished in a variety of ways: aspiration through a needle, by means of a trochar and catheter, by rib resection, and through the pleural space (in one and two stages), through the abdomen; and in very few cases by the extrapleural and extraperitoneal route advocated by Ochsner.

In 1 of the 2 cases of lacerated liver, repeated aspiration through the ninth and tenth intercostal spaces transversing the right pleural cavity effectually emptied a subphrenic collection of cloudy bile-stained fluid which was sterile on culture.

Trochar drainage was used in 6 cases. One was amebic in origin, and it is to be noted that this type of lesion cleared up under various methods of treatment and did not give rise to a pleural empyema when the pleura was contaminated. Apparently this type of abscess is generally free of bacteria. This is in accord with the excellent results at present obtained by the combined use of emetine and multiple aspirations in amebic liver abscess. Another case, effectively treated by trochar and catheter drainage, was one in which an abscess was found a year after a perforated appendix had been removed. The success of this type of treatment was possible in this instance probably because of two factors. The pus had become sterile and the costophrenic angle of the pleura had probably become obliterated. No empyema followed. A third case developed a pleural empyema after the use of a trochar; and a fourth died four days after its insertion.

Aspiration through the pleural space as a diagnostic procedure quite obviously gave rise, in a number of instances, to pleural infection. Instillation of iodine or mercurochrome in the needle tract did not seem to prevent such infection.

Ten cases were drained through the abdomen. Five (50 per cent) died. In 4 of these, pus was spilled into the general peritoneal cavity at the time of the operation. All but 1 were drained through a rectus incision.

The great majority were drained through the pleural space following rib resection. This included 19 cases, of which 7 (36 per cent) died; 7 others (36 per cent) developed a pleural empyema as a complication of the treatment. Of those terminating fatally, 6 probably had pleural infection. Thus, in relatively few cases was it possible to enter the pleural space without causing contamination of sufficient extent to involve the necessity of subsequent drainage, or even the possibility of early death. Suturing the pleural surfaces and using a two-stage transpleural approach did not prevent pleural contamination.

The method of drainage employed by Ochsner was used in only 3 cases of this series. In this approach the patient lies on the sound side; the twelfth rib is resected subperiosteally and the abscess cavity over the liver entered by gentle dissection—the diaphragmatic peritoneum being stripped away from the diaphragm. Neither the general peritoneal cavity nor the pleural cavity is entered. Local anesthesia may be used. An additional advantage of rib resection at this site is the possibility of employing an aspirating needle under direct vision to locate and drain a subhepatic abscess.

For an abscess located anteriorly an incision may be made immediately below and parallel to the costal arch down to the peritoneum. Then, gently stripping the peritoneum away from the under surface of the diaphragm, entry is gained to the pocket of pus without contamination of the general peritoneal cavity. There is much less shock to such a procedure in which neither the pleural nor peritoneal cavity is entered.

In conclusion, the three factors to be emphasized in reducing the high mortality of subphrenic abscess are:

1. Earlier diagnosis—keeping in mind the occurrence of subphrenic infection after perforated ulcers and appendices.

2. Earlier drainage—when the diagnosis is made, drain without delay.

3. A more frequent use of the extrapleural and extraperitoneal approach for purposes of drainage.

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URETHRAL URINARY EXTRAVASATION

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IT IS GENERALLY conceded that the mortality from the extravasation of urine is quite high. Statistics of larger groups of cases give the average mortality at 40 to 43 per cent.[1-3] In our series of 15 consecutive cases occurring in the past ten years, there was only 1 death (6.6 per cent).

ETIOLOGY

It is interesting to note that urinary extravasation rarely results from complete rupture of the urethra. One prerequisite for urinary extravasation, therefore, may be a urethra which is uninterrupted except for one small break. In 5 cases of complete rupture of the urethra seen by the authors, there was no instance of extravasation, presumably because of loss of the desire to urinate or of the expulsive force; consequently urine is not forced into the tissues.

In most instances extravasation occurs only in the presence of a stricture of the urethra, although a few cases have been reported in which there was no stricture. These strictures are, in the main, of gonorrheal origin, but they may be traumatic. Traumatic stricture was present in 5 of our 15 cases. This means that urinary extravasation is a more frequent complication of traumatic than of gonorrheal strictures.

TABLE I

ETIOLOGY OF EXTRAVASATION

Gonorrheal stricture		8
Traumatic stricture		5
Trauma following sounds		2
8 colored	7 white	
Average age		45 years

The beginning of urinary extravasation probably occurs in an infected gland or pocket of scar tissue in the urethra resulting from some old infection or injury located proximal to a stricture. This pocket increases in size as a result of repeated straining in order to void until its wall becomes paper thin. When rupture of this wall finally occurs, urine escapes into the surrounding tissue with each micturition. The amount of urine escaping probably varies with the size, location, and shape of the opening and with the severity of the stricture. In some cases the urine escapes very slowly and leads to a condition resembling

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elephantiasis of the scrotum, as described by Harry Rolnick.[4] Sometimes extravasation has resulted from the dilatation of a urethral stricture either at once or after an interval. Two such cases are included in this series. The etiology of extravasation as seen by us is summarized below in Table I.

Other causes of urinary extravasation from the urethra are the obstruction from a urethral stone, the rupture of a periurethral abscess; in a few cases, occurring in infancy, the etiology is not clear.

DIRECTION OF EXTRAVASATION

Since 66 per cent of urethral strictures involve that portion of the urethra beginning three-fourths inch behind, and ending one inch in front of the triangular ligament,[5] it is natural that most cases of urinary extravasation should have their origin here. In fact, urinary extravasation rarely occurs in any other location. The direction which extravasated urine will take is dependent upon the relationship of the point of rupture to the fascial planes of the surrounding fascia.

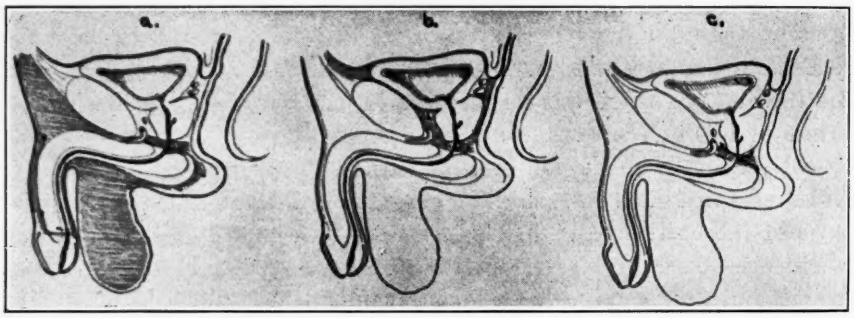

Fig. 1.—Lateral view showing direction of urinary extravasation in the perineal, pelvic, and intraligamentous types (from Meredith Campbell). (*a*) When lesion is anterior to the triangular ligament, a course bounded by Colles' and Scarpa's fasciae is pursued. (*b*) When extravasation occurs on the pelvic side of the triangular ligament, retroprostatic, perirectal, and ischiorectal infiltrations are most commonly observed, although perivesical and prevesical involvement may occur. (*c*) If the extravasation originates within the triangular ligament, extension occurs in either direction, most often externally.

Urinary extravasation from the urethra is usually classified according to its origin as: (1) penile, within Buck's fascia; (2) perineal, anterior to the triangular ligament; (3) pelvic, posterior to the triangular ligament (either into the ischiorectal fossa or subperitoneal space); and (4) intraligamentous, within the triangular ligament.

Extravasation within the confines of Buck's fascia is uncommon and is due to a rupture in the penile urethra. In these cases the swelling is limited to the penis alone. The extent of Buck's fascia and the appearance of such extravasation are illustrated by Miley Wesson.[6]

In the perineal type of urinary extravasation the point of rupture is anterior to the urogenital diaphragm (triangular ligament). This means that the extravasated urine will be limited by Colles' fascia (superficial

perineal fascia), the reflected portion of which forms the superficial layer of the urogenital diaphragm. In front of this layer of Colles' fascia is the superficial perineal pouch which is enclosed by the union of the deep and superficial layers of Colles' fascia in front of the anus. Both layers of Colles' fascia are firmly attached to the ischiopubic rami and ischial tuberosity, but, its superficial layer covers the scrotum and penis, and extends up over the abdomen to become continuous with the deep layer of the superficial fascia of the abdomen (Scarpa's fascia). In the perineal type of urinary extravasation the original swelling in the perineum soon extends to the scrotum and penis, and later over the

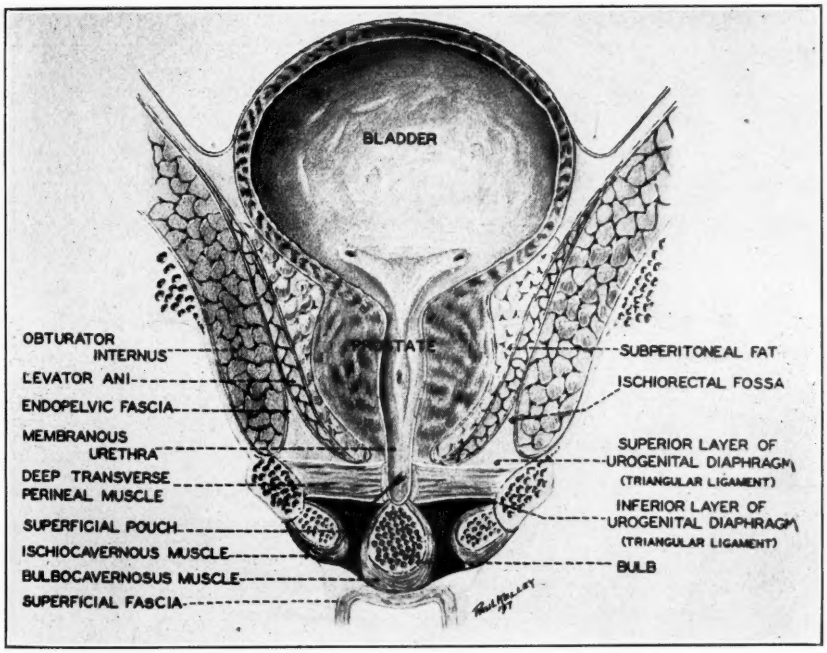

Fig. 2.—Frontal view showing direction of urinary extravasation in the perineal type.

abdomen, in some cases as high as the axillae. Extension into the thigh is prevented by the attachment of Scarpa's fascia to the inguinal ligament. Figs. 1 *a* and 2 illustrate the direction of the perineal type urinary extravasation.

In the pelvic type of extravasation the point of rupture of the urethra is posterior to the urogenital diaphragm (triangular ligament). The deep layer of the urogenital diaphragm is formed by the inferior layer of the endopelvic fascia and forms the floor of the ischiorectal fossa. Anteriorly there is a definite recess extending well toward the body of the pubes and posteriorly there is a recess below the lower edge of the gluteus maximus. Rupture may occur also into the subperitoneal space from which urine may later break into the peritoneal cavity.

In the pelvic type of urinary extravasation there is at first a swelling in the perineum and, as a general rule, extension into the suprapubic recess. Figs. 1 *b* and 3 illustrate the direction of urinary extravasation in the pelvic type.

The intraligamentous type, which follows rupture of the urethra between the layers of the urogenital diaphragm (Fig. 1 *c*), is generally considered to have a better prognosis than the other three since the extravasation is often confined within the layers of the diaphragm.[7] However, it may rupture, either externally or internally, producing either the pelvic or perineal type, or subcutaneously with extravasation into the gluteal region.

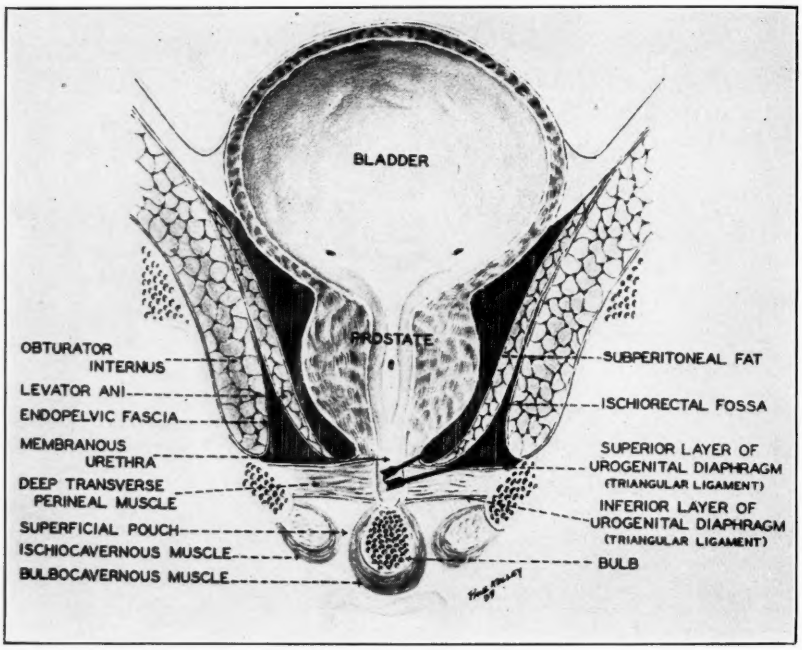

Fig. 3.—Frontal view showing direction of urinary extravasation in the pelvic type.

In our series 11 were perineal, 3 were pelvic, and 1 was pelvic and later perineal. The last may have been an instance of rupture between the layers of the urogenital diaphragm. There was no case of extravasation within Buck's fascia, although most of the perineal type had considerable swelling of the penis.

SYMPTOMS AND SIGNS

The patient complains of a sudden sharp, spontaneous pain, usually in the perineum, followed by the development of swelling and increasing difficulty in urinating. The location of the swelling depends upon the location and size of the rupture and, to some extent, upon its duration.

In most instances there has been previous difficulty in micturition, due either to a gonorrheal or to a traumatic stricture. When first seen, the patient generally feels quite ill, and there may be complete urinary retention.

On examination, the affected parts are at first greatly enlarged and quite tender to the touch. The patient appears toxemic and may have chills and fever. The pulse is somewhat faster than normal. The white blood count is elevated ranging from 14,000 to 28,000 in the acute stages. The urine when first obtained contains considerable albumin. The N.P.N. and creatinin may be elevated. Later he may exhibit mental confusion progressing to delirium and coma.

As the extravasation progresses necrosis of tissue occurs, particularly in the perineal type. There may be, therefore, darkened areas on the scrotum and the penis if the extravasation has been of sufficient duration. It is probable that necrosis is not due to the irritative properties of urine but rather to the pressure of the urine which cuts off the blood supply of the tissues.

TREATMENT

These patients are usually desperately ill and require prompt treatment. Their general condition has often been impaired by the antecedent urinary difficulty with its attendant loss of rest and backflow of urinary products. In addition, the patient with urinary extravasation has increased difficulty in urination from the extravasation itself, as well as imminent or established uremia, and also a severe toxemia from the absorption of the products of degenerating tissue.

Free drainage of the bladder should be provided and adequate incisions for release of extravasated urine and of necrotic tissue should be made. It is better not to waste time in attempting to dilate the already injured urethra. To do so may add just the amount of trauma necessary to make the ending fatal. The perineal cystotomy advocated by some does not seem logical, since the insertion of a catheter into the

TABLE II

TREATMENT OF EXTRAVASATION

Suprapubic cystotomy		8
Cystotomy only	0	
Cystotomy and scrotal incision	5	
Cystotomy and suprapubic incision	3	
Catheter drainage		7
Internal urethrotomy and incision	1	
Sounds and scrotal incision	4	
Sounds and spontaneous rupture	2	
	15	
Deaths	1	
Recovered	14	

bladder must further damage already injured and infected tissue. Good judgment as to the type of procedure to be employed is a prime requisite for the proper treatment of urinary extravasation. The therapy employed in our 15 cases is summarized in Table II.

Discussion.—It may be that the better results which we present are due to the fact that the patients were seen earlier than are those in the hospitals of larger cities where derelicts abound. Certainly few are ever seen in private practice. In this group only 2 were private patients.

SUMMARY

Urinary extravasation from the urethra is accompanied by profound toxemic symptoms from the absorption of the products of damaged tissue and is often accompanied by increasing uremia because of urinary obstruction. The condition is readily recognizable and can be divided into penile, perineal, pelvic, and intraligamentous types. Of these the perineal is by far the most common. The mortality is usually high. Prompt treatment by means of diversion of the urine and by adequate incisions for the extrusion of dead and dying tissue is necessary. Fifteen cases are reported with one death.

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CONCERNING RENAL FUNCTION AND STRUCTURE FOLLOWING TRAUMA OF THE KIDNEY

An Experimental Study*

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THE modern principles of renal surgery demand the preservation of function and the restoration of structure in a diseased organ. The experimental studies of renal function herein recorded, correlated with the pathologic changes secondary to direct injury, are offered as a rational basis for the same intelligent conservatism in the treatment of traumatic lesions of the kidney.

STUDIES OF RENAL FUNCTION†

These experiments were divided into two series: the first was designed to determine the effect of renal trauma on the function of the single kidney of rabbits previously subjected to unilateral nephrectomy; the second, to determine the effect of severe unilateral trauma on the renal function of normal animals and the ability of the injured kidney to recover by subsequent removal of the untraumatized organ.

FIRST SERIES

Method.—Male rabbits weighing between 3¾ and 4¾ pounds were used. No food or water was given after five o'clock in the afternoon of the day preceding the experiment.

The normal renal function of the intact animal was first determined as follows:

The rabbit was placed in a supine position on the animal board. Blood for determination of the nonprotein nitrogen was collected by cardiac puncture. The prepuce was then retracted and a sterile soft rubber catheter, size No. 10 F., was passed into the bladder. The urine was aspirated with a syringe and put aside for later routine analysis. The bladder was irrigated with tap water until the return was clear; the catheter was clamped and left in place.

One hundred to one hundred and fifty cubic centimeters of normal saline solution (depending on the size of the rabbit) were given by gastric tube in order to stimulate a satisfactory flow of urine. One cubic centimeter of phenolsulphonephthalein, containing 6 mg. of the dye, was then injected into a marginal vein of the ear and the exact time recorded at the completion of the injection. The bladder was aspirated through the catheter every ten minutes during the first hour and at half-hourly intervals during the second hour. At each aspiration, the bladder was irrigated with a fixed amount of tap water in order to wash out all the dye therein contained.

*Read at the Annual Meeting of the Halsted Club, Philadelphia, Pa., Dec. 5, 1936. Received for publication, Sept. 28, 1937.

†Two clinical cases and part of these experimental data have been reported in a previous paper.[1]

Each specimen was alkalinized with sodium hydroxide, diluted, and the amount of dye computed by comparison with a series of commercially prepared standard solutions.

Right nephrectomy was performed with sterile surgical technique under basal anesthesia of urethane, supplemented by ether. At intervals, varying from one week to one month after the immediate convalescence, all the preoperative studies were repeated and closely checked.

The left kidney was then traumatized. The organ was exposed retroperitoneally, delivered from the renal fossa, and allowed to lie on the gauze covering the lumbar muscles and skin at the posterior angle of the incision. The anterior surface of the kidney was struck smartly several times with the rounded end of a Wedgewood pestle, about one inch in diameter.

							P.S.P. Test (Minutes)									
	Urinalysis															
Determination	*Color*	*Reaction*	*Sp. gr.*	*Alb.*	*Sug.*	*Sediment*	*10*	*20*	*30*	*40*	*50*	*60*	*90*	*120*	*Total*	*N.P.N.*
Normal Control	V. Cldy. Yel.	Alk.	1.042	Neg.	Neg.	Phosphates	38	31	10	7	4	2.5	4	2.5	99	40
						Right Nephrectomy										
Normal L. Kid.	Cldy. Yel.	Alk.	1.040	Neg.	Neg.	Neg	40	21	11	6	4	3	3	3	91	44
						Operative Contusion of Left Kidney										
1st P. O. Day	Cldy. Red. Yel.	Neut.	1.013	V.H.T.	Neg.	R.B.C., W.B.C., E.C.	2	3	2.5	4	1.5	1.5	4.5	3.5	22.5	150
2nd Day	Cl. Pink. Yel.	Acid	1.008	H.T.	Neg.	R.B.C., W.B.C., E.C.	7	6	4	3.5	3	2.5	6	6	38	160
3rd Day	Cl. Amber	Acid	1.012	T.	Neg.	R.B.C., W.B.C., E.C. H.C.	7	7	4	3.5	3	2.5	8	7	42	129
4th Day	Cldy. Yel.	Acid	1.002	T.	Neg.	R.B.C., W.B.C., E.C., G.C.	2	3.5	3.5	3	3	3.5	9	5	32.5	79
6th Day	Cl. Yellow	Neut.	1.007	S.P.T.	Neg.	R.B.C., W.B.C., E.C.	18	9	9	6.5	4	4	13	6.5	70	71
8th Day	Cldy. Yel.	Alk.	1.027	Neg.	Neg.	W.B.C., E.C.	33	15	11	7	5	3	9	4	87	43
10th Day	Cldy. Amb.	Acid	1.032	S.P.T.	Neg.	W.B.C., E.C.	33	18	12	7	4	3	7	3.5	87.5	33
14th Day	Cldy. Yel.	Alk.	1.028	Neg.	Neg.	W.B.C.	32	25	12	7	4.5	4	6	3	93.5	39
21st Day	Cldy. Yel.	Alk.	1.030	Neg.	Neg.	Phosphates	40	22	8	7	4	3	6	3	93	36
28th Day	Cldy. Yel.	Alk.	1.032	Neg.	Neg.	W.B.C., E.C.	40	30	11	4	4	3	7	2.5	101.5	54

Table I.—Detailed studies of the function of a single kidney following trauma of moderate severity. (From Powers: New York State J. Med. 36: 1, 1936.)

The contused and bleeding organ was then replaced and the wound closed in layers with fine silk.

The function of this single traumatized kidney was studied daily for four days, again on the sixth, eighth, tenth, and fourteenth postoperative days, at the end of three weeks, and finally at the expiration of one month.

Results.—Six animals were included in this series. Three of them, in which the kidney was subjected to trauma of moderate severity, survived. The detailed studies of one animal are presented in Table I. The determinations before and after removal of the right kidney represent the average of two or more observations; those after contusion of the left kidney are single determinations.

							P.S.P. Test (Minutes)									
	Urinalysis															
Determination	*Color*	*Reaction*	*Sp. gr.*	*Alb.*	*Sug.*	*Sediment*	*10*	*20*	*30*	*40*	*50*	*60*	*90*	*120*	*Total*	*N.P.N.*
Normal Control	Cldy. Yel.	Alk.	1.026	Neg.	Neg.	Phosphates	12	45	20	7	3.5	3	3	3	96.5	37
						Right Nephrectomy										
Normal L. Kid.	Cldy. Yel.	Alk.	1.040	Neg.	Neg.	Phosphates	36	22	15	7	5	3	5	3	96	40
						Operative Contusion of Left Kidney										
1st P. O. Day	Bloody	Neut.	1.016	V.H.T.	Pos.	Loaded ♂ R.B.C.	0.5	0.5	0.2	0.3	0	0	1.5	0.5	3.5	173
2nd Day	Clear Yel.	Acid	1.010	H.T.	Neg.	R.B.C., W.B.C., E.C., C	1.0	0.7	0.7	1.0	0.7	0.5	2	1.7	8.3	240
3rd Day	Dead															

Table II.—Detailed studies of the function of a single kidney following severe contusion and extensive trauma. (From Powers: New York State J. Med. 36: 1, 1936.)

The effect of trauma on the function of the single kidney, as measured by the total excretion of phenolsulphonephthalein and the nonprotein nitrogen of the blood, are presented graphically for the group in Figs. 1 and 2. These indices of renal function returned to normal levels within a period of fourteen days after the injury.

The three rabbits whose single kidney was severely traumatized died on the second and third day after contusion. The urine was grossly bloody; the specific gravity was low; heavy traces of albumin were present; the output was diminished; and in one instance complete anuria occurred. Either none of minimal amounts of phenolsulphone-

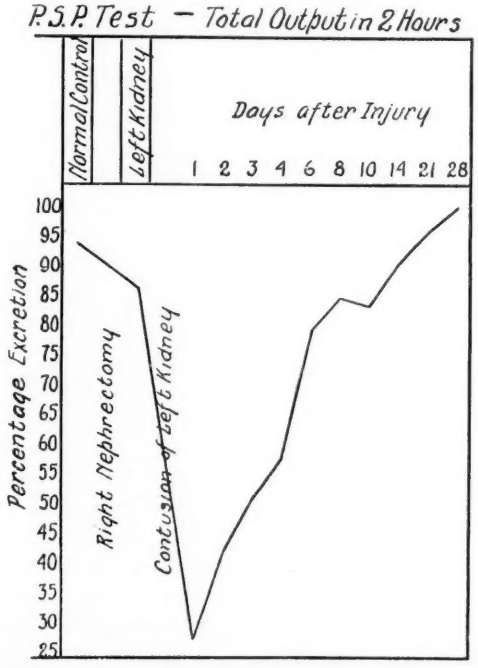


Fig. 1.—Composite curve illustrating the effect of renal trauma of moderate severity on the ability of the single kidney of nephrectomized rabbits to excrete phenolsulphonphthalein. (From Powers: New York State J. Med. **36**: 1, 1936.)

phthalein were excreted and the nonprotein nitrogen of the blood rose rapidly to high levels. The detailed studies of one such animal are presented in Table II.

Conclusion.—Renal trauma of moderate severity impairs the ability of the kidney to excrete urine of normal concentration, reduces the early elimination of phenolsulphonephthalein, decreases the total excretion of the dye, and causes an abrupt and conspicuous rise in the nonprotein nitrogen of the blood. Albumin, red cells, white cells, and casts of various types are found in the urine. The restoration of normal renal function is prompt and complete.

Sudden, severe, and extensive trauma is incompatible with life if the opposite kidney has been removed.

SECOND SERIES

The fatal outcome in the three nephrectomized rabbits whose single kidney was severely traumatized led to the further study of the effect

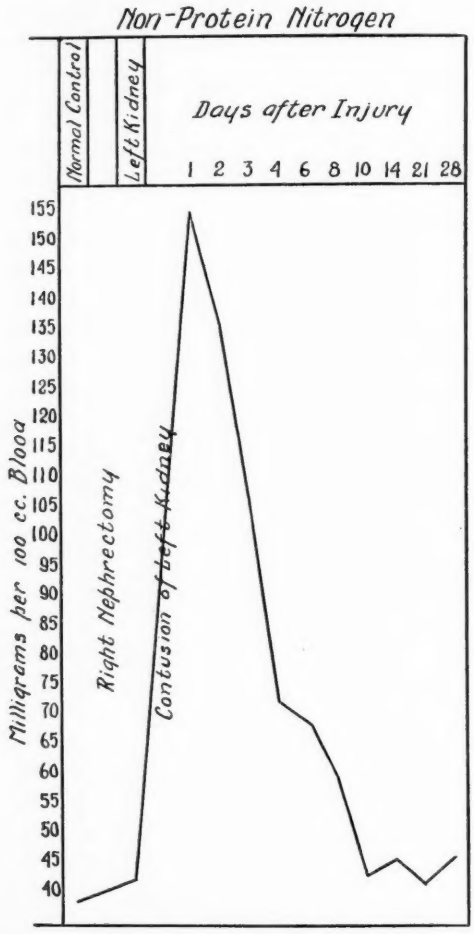


Fig. 2.—Composite curve illustrating temporary nitrogenous retention following renal trauma in rabbits previously subjected to unilateral nephrectomy. (From Powers: New York State J. Med. 36: 1, 1936.)

of extensive unilateral renal injury in rabbits with both kidneys intact. After the immediate post-traumatic period had been safely passed, the undamaged kidney was removed, leaving the animal only the seriously injured organ for the subsequent maintenance of renal activity.

Method.—The preliminary studies were similar to those in the first series. The left kidney was then extensively traumatized by seven to

thirteen blows with the pestle. All the studies were repeated on the first, second, fourth, and eighth days after injury. On the eighth day the normal right kidney was removed. The function of the remaining damaged organ was determined at regular intervals thereafter for one month.

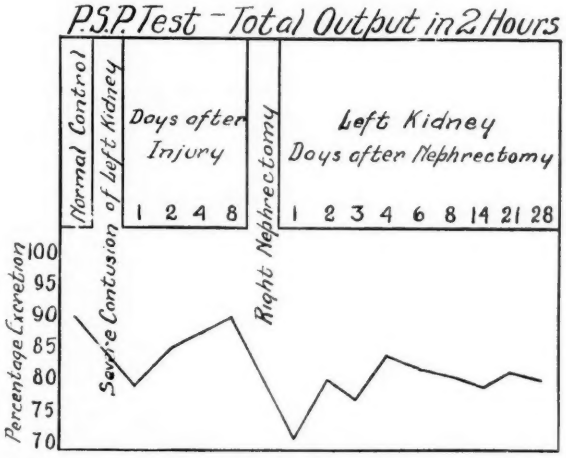


Fig. 3.—Composite curve illustrating the effect of severe unilateral renal trauma on the excretion of phenolsulphonphthalein by normal rabbits; subsequent removal of the uninjured kidney is followed by no significant impairment.

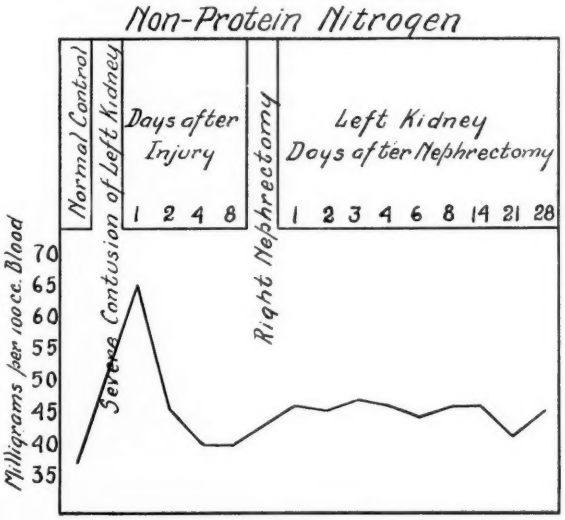


Fig. 4.—Composite curve illustrating temporary nitrogenous retention after extensive injury of one kidney of normal rabbits.

Results.—Nine rabbits were included in the second group of experiments. Two died within twenty-four hours after contusion of the kidney; in each case autopsy revealed extreme distention of the small intestine. Two animals died on the third day, one with pneumonia and one because of persistent, profuse hematuria. One rabbit sustained

RABBIT No. 190

Determination	Urinalysis: Color	Reaction	Sp. gv.	Alb.	Sug.	Sediment	P.S.P. Test (Minutes): 10	20	30	40	50	60	90	120	Total	N.P.N.
Normal Control	Cldy. Yel.	Alk.	1.042	Neg.	Neg.	Phosphates	40	25	9	6	3	2	2	3	90	41
OPERATIVE CONTUSION OF LEFT KIDNEY																
1st P. O. Day..	Cl. Yellow	Acid	1.010	T.	Neg.	R.B.C., W.B.C., E.C.	25	15	10	6	3.5	3	6	3	71.5	100
2nd Day.....	No Urine Obtained						18	17	13	8	6	6	6	6	80	75
4th Day.....	Cldy. Yel.	Alk.	1.028	Neg.	Neg.	Rare W.B.C.	32	15	12	7	6	3.5	5	3.5	84	46
8th Day.....	Cldy. Yel.	Alk.		Neg.	Neg.	Phosphates	15	35	15	7	7	2.5	7	2	90.5	46
RIGHT NEPHRECTOMY																
1st P. O. Day..	Cl. Yellow	Acid		Neg.	Neg.	Few W.B.C.	18	10	8	6	4	4.5	7	4.5	62	60
2nd Day.....	Cl. Yellow	Alk.		Neg.	Neg.	Rare W.B.C.	18	15	8	6	7	4	7	5	70	60
3rd Day.....	Cldy. Yel.	Alk.	1.035	Neg.	Neg.	Few W.B.C.	15	18	10	8	4	5	7	4	71	41
4th Day.....	Cldy. Yel.	Alk.	1.028	S.P.T.	Neg.	Few W.B.C.	20	15	7	6	6	5	10	5	74	54
6th Day.....	Cldy. Yel.	Alk.	1.033	Neg.	Neg.	Carbonates	16	20	7	8	5	4	11	5	76	48
8th Day.....	Cldy. Yel.	Alk.	1.035	Neg.	Neg.	Carbonates	15	18	15	7	7	3.5	10	5	80.5	48
14th Day.....	Cldy. Yel.	Alk.		Neg.	Neg.	Phosphates	20	15	10	6	6	4	8	6	75	60
21st Day.....	Cldy. Yel.	Alk.		Neg.	Neg.	Carbonates	20	15	12	5	5	5	9	4	75	44
28th Day.....	Cldy. Yel.	Alk.	1.019	S.P.T.	Neg.	W.B.C., E.C.	23	17	10	7	5	4	8	4	78	44

Table III.—Detailed studies of renal function following severe unilateral trauma in a rabbit with the opposite kidney intact.

the trauma satisfactorily but died from acute hemorrhage on the day following removal of the uninjured kidney.

The data from the four remaining rabbits are presented graphically in Figs. 3 and 4 for comparison with the first series of experiments, and the detailed studies of one animal are tabulated in Table III. From a study of these figures it is apparent that severe trauma of one kidney of a normal animal may cause only a transient and slight drop in the total excretion of phenolsulphonephthalein or rise in the nonprotein nitrogen of the blood. Following removal of the uninjured kidney, the impairment of renal function is slight.

Conclusion.—If severe injury is inflicted upon one kidney of a normal rabbit, the renal function may be maintained quite satisfactorily by the opposite kidney, with no significant impairment during the immediate convalescent period. If this normal organ is then removed, the traumatized kidney may recover sufficient function to continue the maintenance of renal activity at a satisfactory level thereafter.

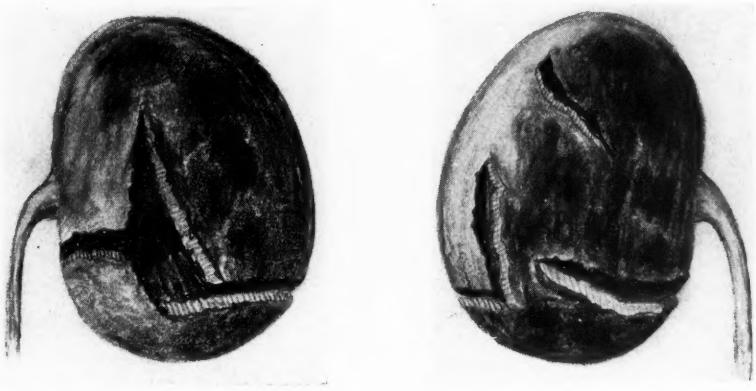
STRUCTURAL CHANGES SECONDARY TO TRAUMA

Gross Anatomic Changes.—Direct trauma of moderate severity (four or five smart raps with the pestle) produced immediate swelling, diffuse subcapsular hemorrhage, capsular tears, and small cortical lacerations on both the anterior and posterior surfaces of the kidney. Severe trauma (seven to twelve blows) caused extensive hemorrhage, ragged wounds of the parenchyma communicating with the pelvis (Fig. 5) and frequently complete decapsulation. The kidney became reddish black in color, soft, pulpy, and lifeless in consistency.

Fig. 6 illustrates the kidney of an animal which was killed on the second day after contusion. The perirenal tissues and overlying peritoneum were edematous and hemorrhagic; the capsule was separated except at the pelvis, shrivelled up, and adherent to the anterior surface

of the kidney. Large, irregular, yellowish gray plaques were apparent superficially and, on section, were found to extend into the cortex for a distance of 1 or more mm. Several clots of blood were found in the pelvis; the mucosa was ruptured in places, and radiating out fan-wise through the medulla were areas of gross hemorrhage.

The return of renal function after injuries of comparable extent and severity was striking.



A. *B.*

Fig. 5.—Operative sketch illustrating the effect upon the kidney of severe experimental trauma. *A*, Anterior aspect; *B*, posterior aspect. (From Powers: New York State J. Med. 36: 1, 1936.)

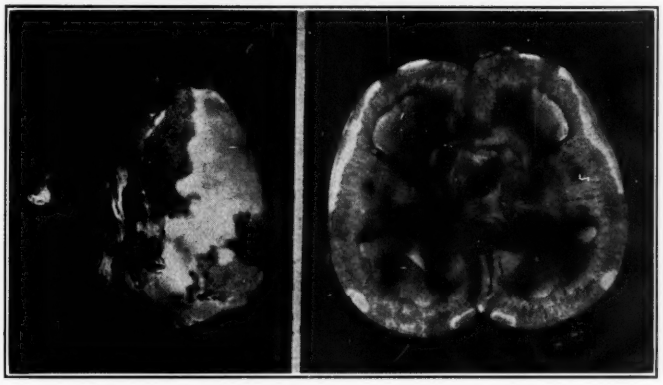


Fig. 6.—Photographs of a kidney removed on the second day after extensive injury. The capsule is attached only at the pelvis; large superficial yellowish-gray plaques and extensive parenchymal hemorrhage are apparent.

Microscopic Changes.—The early histologic changes following injury of moderate severity were those of tubular degeneration with relatively little damage to the glomeruli, greatly swollen and congested vessels, extravasation of blood, cortical hemorrhage continuous with that beneath the capsule, and areas of infarction with complete necrosis of the tubules and infiltration of polymorphonuclear leucocytes (Fig. 7). In

many places the tubules were plugged with hyaline and cellular casts. Mitotic figures were abundant, indicating rapid regeneration of tubular epithelium (Fig. 8).

Fig. 8.

Fig. 7.

Fig. 7.—Photomicrograph of renal infarct showing tubular degeneration and leucocytic infiltration in the infarcted area (×150).

Fig. 8.—Photomicrograph of mitotic figure in tubular epithelial cell (×1350).

Complete destruction of large areas of renal tissue followed trauma of greater severity. Outside these were zones of infiltration with inflammatory cells. Adjacent were areas in which both the degenerating tubules and casts contained large amounts of calcium (Fig. 9). The

histologic picture is not dissimilar to that which is found in poisoning with corrosive sublimate.

Healing occurred by the formation of scar tissue which, in some sections, was very dense, completely replacing all functioning renal tissue. Little blood pigment was left in places of former hemorrhage. No casts were apparent and the changes which remained were those of distortion and cicatrization.

Fig. 9.—Deposits of calcium in degenerating tubules and casts (×150).

Fig. 8.—Photomicrograph of mitotic figure in tubular epithelial cell (×1000).

CONCLUSION

Impairment of renal function following trauma of moderate severity is transient. Severe injury is incompatible with life if the opposite kidney has been removed. Extensive trauma to one kidney, if the other organ is intact, may cause no great change in total renal function. If the undamaged kidney is subsequently removed, the injured organ will regain sufficient function to maintain renal activity at a satisfactory level.

The chief pathologic changes responsible for the transient impairment of function following injury are degeneration of the tubules,

subcapsular and parenchymatous hemorrhage, edema, and infarction. Prompt functional recovery is due to the rapid regeneration of tubular epithelium.

The results suggest that the majority of patients with unilateral renal trauma may be treated conservatively with the expectation that the injured kidney will recover sufficient function to be a useful and serviceable organ.

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NEWER CONCEPTS IN THE TREATMENT OF INJURIES TO THE LIGAMENTS OF THE KNEE JOINT: AN EVALUATION OF THE MAUCK OPERATION

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THE fact, as noted by Jones and Smith,[1] that stable knees may follow severe dislocations of the knee joint treated conservatively; the practice of some surgeons to disregard or excise torn crucial ligaments with satisfactory stable results; and the more recent contributions of several observers who, in the presence of collateral and crucial ligament injuries, have secured stable knees by the repair of the former alone, have strongly suggested the minor rôle played by the crucial ligaments in the integrity and function of the knee joint and, therefore, the lack of need in most cases for severe reconstructive procedures of the Hey-Groves[2] type.

It is our intent (1) to express clinical and experimental confirmation of the above observations, and (2) to emphasize the applicability of the Mauck[3] operation, not only in the treatment of a relaxed internal lateral ligament, but also for the restoration of stability and normal function in a knee joint with associated injuries to the crucial and collateral ligaments.

ANATOMIC CONSIDERATIONS

The tibial collateral (internal lateral) ligament is attached to the medial condyle of the femur, near the adductor tubercle; it crosses the medial aspect of the joint where it becomes attached to the peripheral border of the internal meniscus and is inserted into the medial border of the tibia, behind the semitendinosus tendon. It consists of a long superficial and a short deep portion, and it is the latter which shares the intimate relation with the internal semilunar cartilage.

The fibular collateral (external lateral) ligament is a distinctly palpable, rounded band attached to the lateral femoral epicondyle; it is separated from the lateral aspect of the knee joint by the popliteus tendon and its bursa. At its insertion to the fibular head, the two slips of attachment of the biceps tendon with an interposed bursa embrace it.

The anterior crucial (cruciate) ligament, attached above to the outer femoral condyle far back in the intercondylar notch, is directed downward, forward, and a little inward to its attachment in front of the tibial spine.

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The posterior crucial (cruciate) ligament is attached above to the front part of the inner aspect of the femoral intercondylar notch and passes downward, backward, and a little outward to become attached to the tibia at the posterior portion of its articular surface.

The integrity of the knee joint depends not only on these ligamentous structures but also upon the posterior oblique ligament, the muscular aponeuroses and tendons about the joint, and, to a less extent, the capsule and menisci.

The lateral ligaments become taut in full extension, checking hyperextension, lateral motion, and rotation. In flexion, since their femorotibial attachments approximate, they are relaxed; the knee joint becomes loose, and slight lateral and rotatory motion about a longitudinal axis now becomes possible (Steindler[4]).

The usual conception of crucial ligament function, based on its anatomic construction, is that the anterior crucial ligament, checking forward displacement of the tibia, becomes tense in full extension, while the posterior crucial ligament, checking backward displacement of the tibia, becomes taut in full flexion. Milch[5] notes that this is at variance with the studies of Weber,[6] Meyer,[7] and Fick,[8] who observed that during flexion and extension different portions of both crucial ligaments became taut in different stages of motion, and that the position of maximum tension of both crucial ligaments was that of flexion and internal rotation of the tibia on the femur. "From these considerations it appears reasonable to conclude that the crucial ligaments function mainly during the act of flexion, and that during extension the support of the knee is undertaken by other structures, viz., the collateral ligaments and the muscles about the joint" (Milch).

EXPERIMENTAL CONSIDERATIONS

Giertsen[9] quotes Hyrtl's anatomic observations that (1) in extension the stability of the knee remains intact if the crucial ligaments are severed, provided that the collateral ligaments remain intact; and (2) instability in flexion and extension results if the collateral ligaments are severed and the crucials left intact.

Weber,[6] as noted by Milch, observed that with the collateral ligaments intact and the crucial ligaments severed, the knee was stable in extension and unstable in flexion, while the reverse conditions existed if the collateral ligaments were sectioned and the crucials preserved.

Pagenstecher[10] ruptured the anterior crucial ligament in cadavers by blows from behind applied to the upper end of the tibia with the knee flexed, and the posterior crucial ligament by blows upon the front of the upper tibial extremity. He could rupture the anterior crucial ligament only at its femoral attachment.

Honigschmeid[11] noted that (1) hyperextension produced a loosening of the crucial ligament attachments, of the anterior from the tibia, and of the posterior from the femur; (2) excessive flexion tore the anterior crucial ligament from its femoral attachment; (3) excessive adduction of the leg on the thigh ruptured the external lateral ligament, the anterior crucial ligament, and then the posterior crucial ligament; (4) excessive abduction detached the internal collateral ligament, posterior crucial, and then the anterior crucial ligaments; (5) excessive internal rotation of the tibia on the femur ruptured the capsule and anterior crucial ligament; and (6) excessive external rotation of the tibia detached the posterior crucial ligament from the tibia, ruptured the anterior crucial ligament, and detached the internal meniscus.

Pringle[12] observed that, if through a small incision the anterior crucial ligament was divided, no abduction was possible in the extended position and only a slight degree in the flexed position, but that the range of abduction was greatly increased with section of the internal collateral ligament. He produced rupture of the anterior crucial ligament in cadavers by fixing the pelvis, flexing the knee, abducting the leg and rotating it inwards on the femur. In all cases he noted that there was concomitant tearing of the deep fibers of the internal lateral ligament.

Observations by one of us (M. T. H.) on the knees of ten cadavers at the Daniel Baugh Institute of Anatomy of the Jefferson Medical College and on fresh specimens from four large dogs serve to corroborate many of the above findings. The integrity of the knee was disturbed as little as possible through small anteromedial incisions and the necessary severance performed with a tenotome. It was noted that:

1. With the knee fully extended, section of the anterior crucial ligament, posterior crucial ligament, or both, did not disturb the stability of the knee. No undue lateral or rotatory motion and only very slight anteroposterior motion was present.

2. With the knee in full extension, the collateral ligaments severed, and the crucial ligaments intact, definite anteroposterior, lateral, and rotatory motion existed, which became greatly exaggerated with severance of the crucial ligaments.

3. With the knee flexed, if the anterior crucial ligament was severed, anteroposterior (''rocker'') motion became evident; but this was greatly increased, as was also the range of abduction and rotation of the tibia on the femur with the knee flexed, when the internal collateral ligament was severed.

4. With the knee flexed, even though the anterior crucial ligament was left intact, severance of the internal lateral ligament resulted in definite ''rocker'' motion, with increase in abduction and rotation of the tibia on the femur.

5. In two cadavers, with the pelvis fixed and the knee flexed, the leg was forcefully abducted on the femur. The internal lateral ligament was torn from its tibial attachment in each case, and with the abducting force continued, marked stress was placed upon both crucial ligaments, the greatest strain being exerted on the anterior when the leg was internally rotated and on the posterior when the leg was externally rotated. In this position the internal meniscus, its anterior and posterior attachments intact, became displaced into the knee joint, but no actual tear was seen to occur, possibly due to the absence of superincumbent weight.

CLINICAL CONSIDERATIONS

These anatomic studies serve to confirm the clinical experience of a small group of observers.[13-26] Their contentions are well expressed in the conclusion of Milch's[5] excellent paper: "I have come to believe that, contrary to general opinion, the anterior crucial ligament is not a vitally necessary structure and that its loss is thoroughly compatible with relatively normal function of the knee, that the diagnosis of rupture of this ligament alone is not as simple as it has been stated to be, that the tests ('rocker' and extension tests) are in all probability indicative of injuries to both the crucial and the internal lateral ligaments, that the disability is due primarily to the loss of integrity of the internal lateral ligament, and that in consequence surgical efforts should be directed primarily toward the repair of the internal lateral ligament . . . rather than repair of the crucial ligament. . . ."

The great improvement in the results of crucial ligament repair with fascia or tendon by the Alwyn Smith[13] modification, of additional reconstruction of the internal lateral ligament, was immediately appreciated and accepted by Hey-Groves[27] and utilized by later contributors. Bennett[17] successfully treated five out of six cases with torn anterior crucial ligaments confirmed by arthrotomy by his method of plication of the medial capsule and fascial reenforcement of the internal lateral ligament alone, with resulting normal stability and function. Cotton and Morrison,[21] and Bosworth and Bosworth[23] secured stable knees with their methods of internal lateral ligament reconstruction, disregarding the repair of the associated torn anterior crucial ligaments. Tixier and DeRougemont[24] excised a fractured tibial spine and the entire anterior crucial ligament with resulting excellent stability and knee function, confirming similar experiences by Tavernier,[14] Leriche and De Girardier,[16] and Constantini and Conniot.[25] In discussing this paper Tavernier[26] reported a case of anterior crucial and internal lateral ligament tears, wherein restoration of the latter alone resulted in a normal knee. Milch,[5] also, has noted satisfactory knee-joint stability following the resection of a torn anterior crucial ligament. Most recently, Mauck,[3] in three cases, one with demonstrable tear and two with

relaxed anterior crucial ligaments and each with a positive "rocker" sign and lateral instability, secured stable knees by employing his method of internal lateral ligament reinforcement.

TREATMENT

Injury, either as actual tears or stretching, to the internal collateral ligament should be anticipated in all injuries of the knee joint. Secondary relaxation may follow uncontrolled intraarticular effusion. Conservative therapy is indicated in the early stages, with aspiration deferred until forty-eight hours after the injury is incurred and then repeated if necessary. Immobilization is best secured with a close-fitting plaster bandage, and in injuries of the collateral ligaments alone this may be hinged at the knee to allow flexion and extension and yet inhibit lateral stress and torsion.

For the surgical reconstruction of the internal lateral ligament, Alwyn Smith[13] utilizes the additional length of the fascial strip employed to reconstruct the anterior crucial ligament, to bridge the medial aspect of the knee joint, suturing it to the internal femoral condyle. Bennett[17] plicates the relaxed internal capsule, reinforcing this in his more recent cases with fascial strips. Cotton and Morrison[21] and Bosworth and Bosworth[23] employ a fascial strip, run through drill holes on the medial surfaces of the inner femoral and tibial condyles and made to bridge the joint in cross-fashion. Wilson[28] and Campbell[29] use a pedunculated fascial flap, its femoral attachment preserved, suturing it to the medial surface of the internal tibial condyle. Others prefer the use of adjacent tendons to reinforce the internal ligament; McMurray[30] utilizing the sartorius; Eikenbary,[15] the semitendinosus; Edwards,[31] the gracilis and semitendinosus; and Phillips,[32] the gracilis alone.

Mauck[3] described his procedure for the treatment of relaxation of the internal lateral ligament following trauma and effusion and unrelieved by a conservative regime. He attributes such relaxation to detachment of the internal lateral ligament with its attached internal semilunar cartilage from its tibial insertion. The resulting hypermobility is often aggravated by the removal of the internal meniscus, deranged or benign, and such a knee is predestined for further derangement. His procedure is performed as follows:

The inner aspect of the knee joint is exposed through a slightly anterior curved incision, extending from the adductor tubercle of the femur to 4 inches below the joint line, and the skin and fascia are dissected anteriorly and posteriorly. With a broad chisel the inner side of the tibial head, with the attached internal lateral ligament, is removed and reflected upwards, this triangular-shaped flap measuring 1½ inches in length and ½ inch in thickness at the articular surface. The capsule is incised at the anterior and posterior margins of the bony flap, and the internal meniscus, attached to the capsular flap, is excised.

The knee joint may now be thoroughly inspected. To shorten the internal lateral ligament, the bone flap is pulled downwards forcefully, and with the leg fully extended and strongly adducted it is mortised into a trough prepared ¾ to 1¼ inches below its former site, on the medial aspect of the tibia. Thus the lower end of the ligament, i.e., its weakened fibrous portion, contacts a fresh bony surface to which it becomes adherent (Fig. 1 *A* and *B*). The capsule is plicated if necessary and the subcutaneous tissue and skin closed. Mauck then applies a plaster bandage with the knee in full extension for six weeks, and this is hinged at the knee at the end of two weeks to allow early flexion and extension.*

This technique has been employed by the senior author (A. J. D.) in the treatment of two cases, each with proved anterior crucial ligament tear, torn internal meniscus, and marked relaxation of the internal

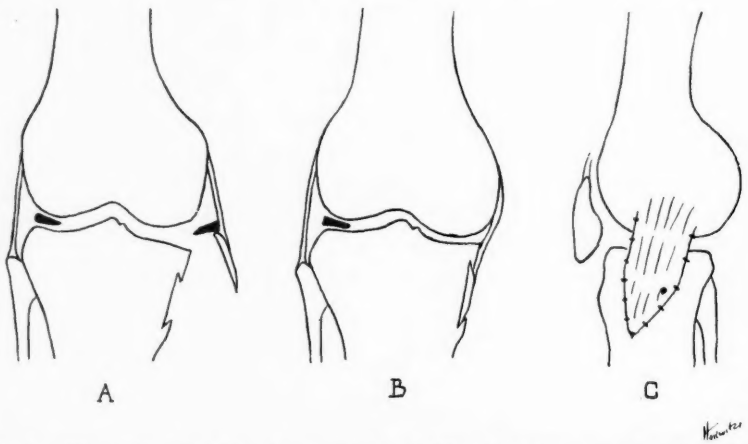

Fig. 1.—Illustrating the Mauck operation. *A*, The tibial bone flap and its attached internal lateral ligament reflected upwards, and a trough constructed on the medial tibial surface; *B* and *C*, the bone flap displaced downwards and forwards under tension and mortised into place following removal of the internal semilunar cartilage.

collateral ligament. The whole bone flap was implanted forward as well as downward to increase the obliquity of the anterior fibers of the ligament and to act as an additional check against forward movement of the tibial head on the femur, as recommended but not practiced by Mauck (Fig. 1 *C*). The procedure seems most applicable for this type of case because it permits an excellent exposure of the interior of the knee joint without unduly disturbing its capsuloligamentous envelope; because the removal of the internal meniscus, deranged or normal, permits a close approximation of the tibia to the femur; and because it is a direct attack upon and actual shortening of the internal lateral ligament rather than a substitution by fascia or tendon.

*Wittek, A. (Zur Wiederherstellungs-Chirurgie des Kniegelenkes, Wien. klin. Wchnschr. 50: 803, 1937) describes an exposure of the knee joint not unlike the Mauck operation. He chisels off a portion of the medial femoral epicondyle, turning it down with a portion of the capsule, and, following repair of the internal structures, nailing the separated epicondyle into a new bed proximal to its original position, under tension.

CASE REPORTS

CASE 1.—W. L., a colored male, aged twenty years, injured his left knee in a fall one year prior to admission, with resulting persistent disability and instability. On examination the left knee evidenced periarticular swelling and a moderate synovial effusion. The range of motion was from full extension to a right angle. There was definitely localized tenderness in the region of the anterior attachment of the internal meniscus. With the knee fully extended, definite anteroposterior motion of the tibia on the femur was elicited and the leg could be abducted 15° to 20°. With the knee flexed, the "rocker" sign was strongly positive. Roentgenographic studies of the affected knee and all laboratory studies were negative.

A preoperative diagnosis of torn anterior crucial ligament, torn internal meniscus, and relaxed internal collateral ligament was confirmed at the time of operation (Feb. 3, 1937), at which time the Mauck procedure was performed. The extremity was immobilized in a plaster bandage for two weeks and hinged at the knee for four additional weeks.

Restoration of function was rapid with complete return of motion six weeks following the removal of the plaster. All anteroposterior and abnormal lateral motion had disappeared. To date there has been no secondary relaxation and the patient has returned to his vocation of bell-hop.

CASE 2.—L. K., a white male student, aged eighteen years, injured his left knee one year prior to admission while playing ball. He thereafter suffered persistent pain, swelling, weakness, and instability. Examination revealed the left knee to be moderately swollen due to a synovial effusion and thickened synovial membrane. There was localized tenderness at the anterior horn of the internal semilunar cartilage and in the region of the tibial attachment of the internal lateral ligament. There was undue anteroposterior and lateral motion of the tibia on the femur with the knee fully extended and a positive "rocker" sign with the knee flexed. The range of motion was normal. X-ray studies of the injured knee were negative.

A preoperative diagnosis of torn anterior crucial ligament, torn internal meniscus, and relaxed internal collateral ligament was confirmed at operation (June 20, 1937), at which time a typical Mauck procedure was performed. The knee was completely immobilized in plaster bandage, in full extension, for three weeks.

Normal function returned rapidly following the removal of fixation. The abnormal anteroposterior and lateral movements were no longer evident and the "rocker" sign was barely elicited. There has been no secondary relaxation up to the present.

In two other cases with disruption of the anterior crucial ligament, corroborated at the time of arthrotomy for deranged internal menisci, but with intact internal lateral ligaments, excellent stability without any preternatural mobility resulted, although the crucial ligament tears were disregarded. One other case is significant, a severe, traumatic, medial dislocation of the tibia on the femur with unquestionable severance of both crucial ligaments. Conservative treatment was instituted and the extremity encased in a plaster bandage, with resulting excellent stability and normal function. We feel that in this case the internal collateral ligament was not critically ruptured and possibly only severely stretched.

CONCLUSIONS

1. A review is given of previous experimental and clinical observations and our own contributions are described to substantiate the con-

cept that the anterior crucial ligament is not vital to the integrity of knee joint stability and function; that the diagnostic signs usually described as indicative of crucial ligament injury are also indicative of internal collateral ligament derangement; and that surgical procedures should seek to correct the relaxed or torn internal lateral ligament alone.

2. The Mauck procedure is described and its value in the correction of relaxation of the internal lateral ligament is corroborated.

3. The adaptability of the Mauck technique for the repair of the relaxed internal lateral ligament associated with tear of the anterior crucial ligament is stressed. Where the internal ligament is frankly and irreparably ruptured, a fascial or tendinous reconstruction becomes obligatory.

We are deeply grateful to Professor J. Parsons Schaeffer, Director of the Daniel Baugh Institute of Anatomy, Jefferson Medical College, for the liberal supply of anatomic material.

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POSTERIOR DISLOCATION OF THE FIRST CERVICAL VERTEBRA WITH FRACTURE OF THE ODONTOID PROCESS

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THE incidence of dislocation of the upper cervical spine associated with fracture is not rare. In fact, the literature abounds in numerous reports of the condition. In looking through the literature, however, one is impressed with the absence of posterior dislocation of the first cervical vertebra and the treatment of this apparently unusual injury. This is the reason for reporting the single case, the report which is as follows:

CASE REPORT.—Mrs. L. M., aged fifty-five years, was riding in the front seat of an automobile next to the driver on Oct. 27, 1934, when she was thrown out of her seat in a head-on collision, her forehead striking the sill above the windshield and her head being thrust forcibly backward. She immediately sensed increasing difficulty in using her arms and was helpless when she arrived in the hospital. She states that she was numb from her neck down; and whereas she had some control of her arms and legs, she was unable to regulate these movements, which were more or less purposeless. She sustained a very extensive scalp wound involving almost the entire frontal and biparietal areas. Her scalp wound was sutured and she was placed in bed with sandbags on either side of her head.

She was seen by me in consultation ten days after the accident. At this time, examination revealed an extensive suppurating scalp wound involving the frontal and biparietal regions. The chin was pointed upward and to the right. There was complete limitation in rotation of the head, and the other movements of the cervical spine were markedly restricted. The pronator, biceps, triceps, patellar, and Achilles reflexes were all present and symmetrical, but markedly increased, slightly more on the left side. She stated that since her accident she had noticed a slight improvement of her right arm, so that at the time of my first examination she was able to grasp an object on the bedside table next to her. The left arm, however, was "still entirely out of control." She was able to move her lower extremities and there was no sensory disturbance. There was no urinary or fecal incontinence.

Roentgenograms (Figs. 1 and 2) taken immediately after the accident showed the first cervical vertebra to be dislocated posteriorly for a distance of approximately ⅓ inch, and to the left side for a distance of approximately ¼ inch. Obviously, the articular facets were also dislocated and there was a fracture of the odontoid process which was displaced posteriorly.

The patient was removed to Mt. Sinai Hospital, Cleveland, Ohio, where reduction was attempted as follows: The patient was given preliminary hypodermic injection of morphine sulphate, gr. 1/6, and atropine sulphate, gr. 1/150, followed by tribromethanol anesthesia, 80 mg. per kilogram of body weight. She was then placed on the Albee fracture table and reduction was accomplished by reversing the Walton maneuver. In other words, the patient was placed supine instead of prone on the fracture table and a halter was applied and fixed to a web strap which was placed around my waist. Because of the necessity of obtaining an upward pull, it was necessary to be elevated on a stool, approximately eighteen inches high. By

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means of constant pull obliquely upward, the head was turned first to the left side, and then to the right. There was no click as frequently occurs in anterior disloca-

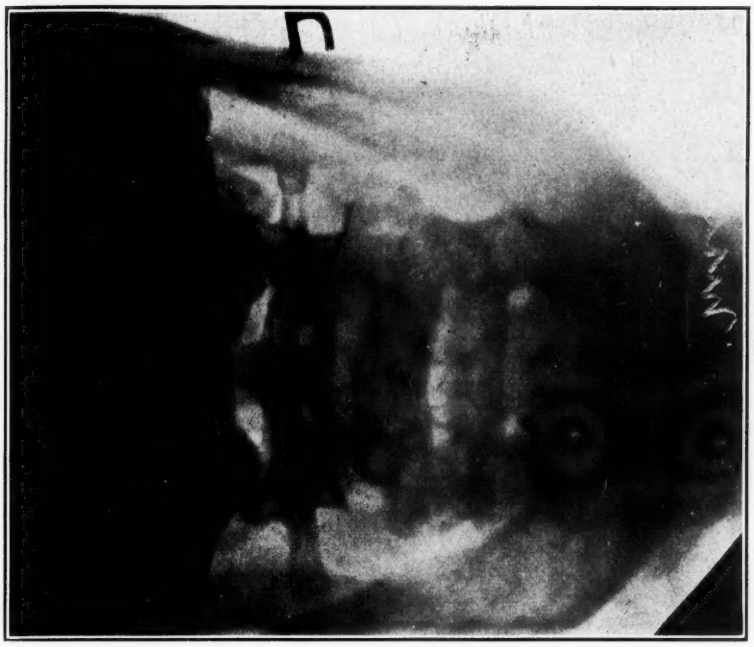

Fig. 2.

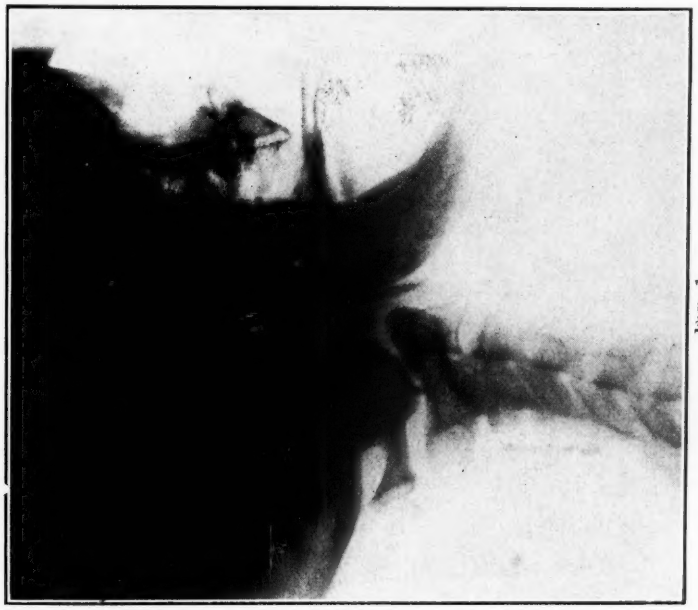

Fig. 1.

Fig. 1.—Lateral view showing posterior dislocation of the first cervical vertebra on the second, with fracture of the odontoid process displaced posteriorly.

Fig. 2.—Anteroposterior view showing dislocation of the articular processes between the first and second cervical vertebrae to the left.

tions; in fact, there was no sensation whatever of a reduction. The head was maintained in this position until another roentgenogram was obtained by a portable unit. This showed a complete reduction as illustrated in Figs. 3 and 4. In this

Fig. 3.—Lateral view showing reduction of dislocation of the first cervical vertebra and alignment of the fractured odontoid process.

Fig. 4.—Anteroposterior view showing reduction of the laterally dislocated articular processes. The odontoid process is seen fractured at its base.

position, a plaster of Paris bandage was applied, including the upper torso, shoulders, neck, and head, cutting out a window for dressing the scalp wound, and the patient was placed in bed.

Within three days she began to note improvement of the sensory function of her left arm, and after approximately ten days, the motor control of the left arm became evident, although the movements at that time were still incoordinated. However, within approximately another two weeks the patient was able to reach for articles and grasp them, and from then on motor recovery continued to improve. About two months after reduction, she was able to feed herself. Four weeks following the reduction and application of the cast, it was removed and another plaster bandage applied, which now included the entire scalp, as the scalp wounds had healed. This remained applied for approximately three and one-half weeks, when it was removed and replaced by another. This bandage remained applied for six weeks, after which period it was removed and an aluminum neck brace applied. This brace was removed periodically and active rotation encouraged.

Three months following the reduction, the patient was able to get about without any difficulty whatever and had complete control of her right arm, although she still complained of some "weakness" in her left. This weakness did not impair her from using it for the usual necessities. The reflexes of the arm and forearm had diminished in intensity and were now symmetrical. In spite of the fact that the patient had no difficulty with the lower extremities, the patellar reflexes continued to be slightly exaggerated up to the last examination, Dec. 17, 1936.

After six months the brace was entirely discarded and more active rotation encouraged. Rotation is now approximately 75 per cent of normal, and the other movements are not impaired.

COMMENT

1. Search of the literature reveals that many cases of dislocation of the first cervical vertebra associated with fracture have been reported, but these cases are all anterior dislocations. Brooks' various articles are probably the most comprehensive, notably, the one published in 1933 which reports forty cases, of which twenty were fractures of the first cervical, every one being an anterior dislocation. These anterior dislocations present a very definite deformity; namely, the head tilted forward, with the chin on the upper thorax in some instances. In the posterior dislocation, the head is obviously in the opposite position, being tilted backward with the head a little to one side and the chin pointing upward.

2. It is interesting to note that the Walton maneuver also may be applied to the posterior type of dislocation, with position of the patient and the maneuver naturally reversed. This type of maneuver has been very successful in this case.

3. I have been particularly gratified with the use of rectal anesthesia in this case, as it worked perfectly. The patient was always under control, and we did not experience any inconvenience at having the anesthetist in our way, which would necessarily be the case in inhalative anesthesia. This method also has been used numerous times by the writer in reducing compression fractures of the spine.

SUMMARY

A case of posterior dislocation of the first cervical vertebra associated with fracture and also with fracture of the odontoid process of the second cervical vertebra is reported. The Walton method of reduction, reversing the position of the patient and reversing the maneuver, proved very successful. Rectal anesthesia, by the employment of tribromethanol, offered complete relaxation without the inconvenience of having an anesthetist in one's way.

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RECTOVAGINAL CLOACA

Report of a Case and a Simple Method of Reconstruction

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SINCE Bodenhamer's definitive volume on congenital malformations of the rectum and anus, which was written in 1860, there have been few published instances of the surgical treatment of rectovaginal cloaca. A notable exception to this was the description by Stone of his operation for this defect in 1936. His procedure is suited for the cases in which the rectum opens into the posterior wall of the vagina at a variable distance above the vaginal introitus. In our case the rectum opened into the vagina at the posterior fourchet. This, according to Stone, is the commonest type of rectovaginal cloaca. It seemed advisable, therefore, to report the case in which we recently performed an operation by a simple method and in which the result was successful.

From the standpoint of comparative anatomy, the cases reported by Stone represent an arrest at an earlier stage in the evolutionary scale than does our case. In amphibia the rectum opens into the top of the cloaca, which would correspond to an opening of the rectum into the posterior fornix of the vagina of the malformed human female. The opening of the rectum passes downward as one ascends the evolutionary scale to the reptilia, in which it opens midway down the cloaca. In marsupials the rectum opens into a very small cloaca very near the perineum. This corresponds to the condition obtaining in our case.

Sir Arthur Keith said that these malformations occur in one of about every 5,000 infants so it may be beyond the experience of any one physician to encounter the condition. Yet, if the condition is encountered, one should know a safe and successful method of treatment. It is the consensus that operative treatment should not be instituted until the child reaches the age of at least six or seven years. Spontaneous separation of the rectum and vagina has occurred when operation has been thus postponed. Dilation of the vaginal orifice of the rectum is usually sufficient, as a temporary expedient, to permit the patient to have unimpeded bowel movements until operation is deemed advisable.

Lynch and Hamilton have recently described congenital abnormalities of the rectum and suggested operative procedures for their correction.

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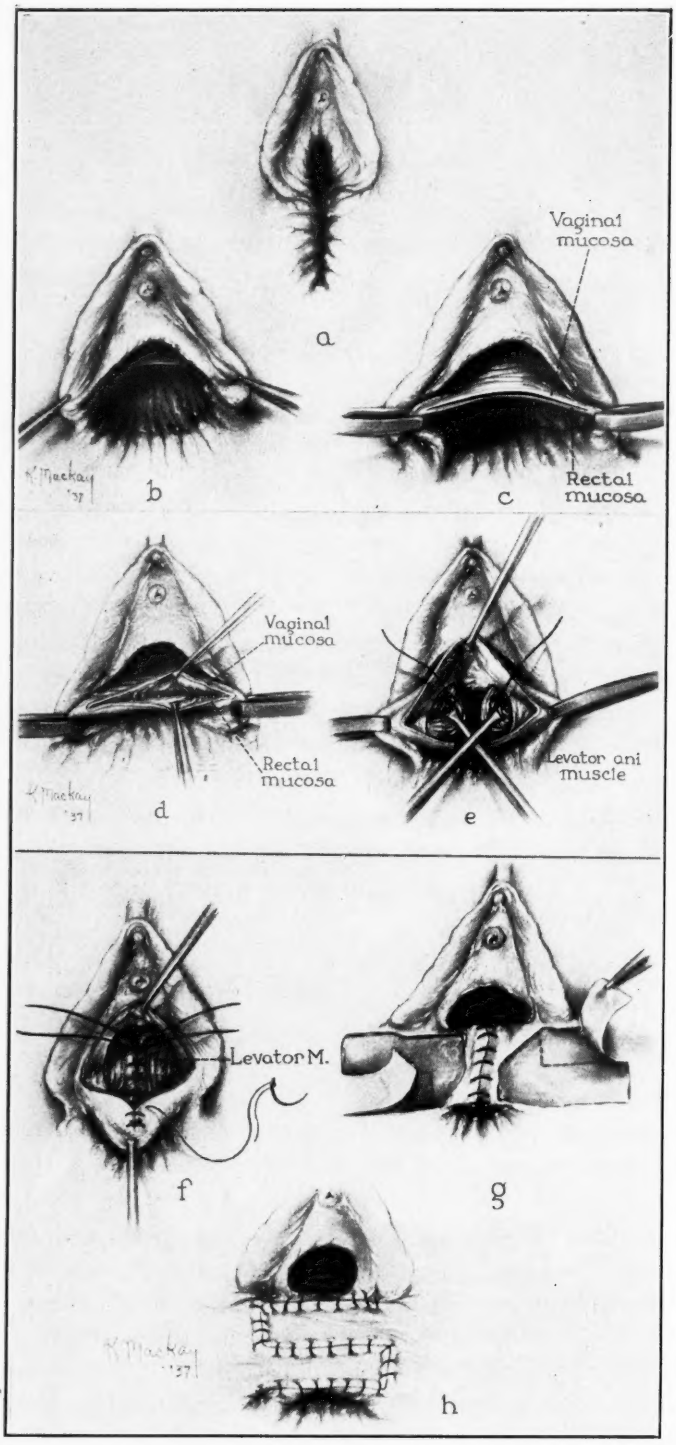

Fig. 1.—Repair of rectovaginal cloaca; *a*, condition of patient before operation; *b*, the labia are parted and upper part of incomplete rectovaginal septum can be seen; *c*, incomplete rectovaginal septum has been pulled down with tenaculum forceps and incision made in its free border; *d*, separation of the rectal and vaginal mucous membranes; *e*, dissection and isolation of the levator ani muscles; *f*, levator ani muscles are approximated in the midline; *g*, reconstruction of perineal body; *h*, use of skin flaps for plastic closure.

REPORT OF CASE

A girl, aged eleven years, was brought to the clinic because her "rectum was not in the right place." Shortly after her birth her parents had noted that the rectum opened into the same cavity as did the vagina. The opening was small and feces had to be extracted manually on numerous occasions. When she was one year old her physician had dilated the opening of the rectum and this had facilitated her bowel movements considerably. However, she had continued to depend on the frequent use of cathartics and enemas. Three years before her admission to the clinic she had been advised to take mineral oil daily, which had aided in the expulsion of feces. During the year before the patient came to the clinic her sphincteric control had been poor and there had been daily soiling of her clothes.

The urethral orifice was normal and the vagina was well developed. The lower half of the rectovaginal septum was entirely lacking. The rectum communicated with the vagina at this point. The fourchet had been replaced by the posterior rectal wall. The anterior half of the external and sphincter was absent, but a semicircular posterior portion was palpable and partially surrounded the posterior rectal wall.

Proctoscopic examination revealed that the rectum and sigmoid colon were dilated to two or three times their normal diameter throughout the distal 24 cm. A barium enema was given, but the roentgenogram of the colon was unsatisfactory because of the patient's inability to retain the enema. However, the roentgenologist did not find any evidence of a megacolon.

The operative method used was an adaptation of the commonly used means of restoring the perineal body following laceration caused by childbirth. In this instance it has served very well to construct the perineal body of a girl who had never had one. Fig. 1 *a* shows the condition of the patient when she presented herself for examination. The posterior half of the external sphincter of the anus was present and could be palpated. By parting the labia, the upper part of the incomplete rectovaginal septum could be seen (Fig. 1 *b*). This was pulled down with tenaculum forceps and an incision was made in its free border (Fig. 1 *c*). The vaginal and rectal mucous membranes were then carefully dissected back (Fig. 1 *d*) until the edge of the levator ani muscles could be located and grasped on each side (Fig. 1 *e*). The levator ani muscles were sutured together in the midline with interrupted sutures of chromic catgut (Fig. 1 *f*). The submucous tissue was then united over the approximated muscles (Fig. 1 *g*). Finally, skin flaps were raised laterally and slid over across the newly constructed perineal body (Fig. 1 *h*).

The wound healed by first intention without any drainage. The patient was able to control her bowel movements perfectly without any soiling of the clothes. When she was dismissed from the hospital, the anus was separated from the vagina by 1½ inches (3.7 cm.) of skin, and a firm rectovaginal septum was present.

COMMENT

A recent letter, which expressed the happiness of the parents and the patient with the result of the operation, further attests to the efficacy of the operative procedure.

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Editorials

Opportunities for Graduate Training in Surgery

THE American Board of Surgery has shouldered upon the surgeons of our country a great responsibility. The qualifications demanded for examination by the Board specify certain periods of training in hospitals and medical schools after receiving the degree of M.D.: (1) an interneship of not less than one year, (2) further training in surgery in hospital and medical school of not less than three years, (3) adequate operative experiences in which the candidate has assumed the whole responsibility, and (4) an additional period of not less than two years of study or practice in surgery. The natural corollary to these demands is that there be places where a prospective candidate may secure this experience. Are there such places and are there a sufficient number of such places to educate all the men who should have and would like to have this experience?

After a personal survey of a large number of medical centers and hospitals and after a careful study of the very valuable data compiled each year by the American Medical Association and published in the hospital or educational numbers of their journal, I am convinced that the opportunities now afforded to fulfill the qualifications demanded by the American Board of Surgery are inadequate.

The material published for many years by the American Medical Association in the hospital number of the Journal is of considerable value to the young medical graduate in choosing the hospital to which he may go for further education. These tables, however, do not contain lists of posts beyond the interneship, nor do they specify the percentage of free beds nor the autopsy percentage, which are prime criteria for the student to study if he is to choose a hospital where his experience will be considerable and instructive. In addition to the information in this number, the American Medical Association has published in their educational number for many years data concerning hospitals, at first listing hospitals approved for interneships by the Council on Medical Education and Hospitals of the American Medical Association. In the last five years it has published a separate list of hospitals approved for residencies as well. The lists of such approved residencies in the educational number of the *Journal of the American Medical Association* are not of great value preceding 1934, because in the years antecedent to that time the list contained merely the location

of the hospital and its bed capacity. Beginning in 1934, there was added considerable other information of assistance to prospective candidates, such as the percentage of free and pay patients, the percentage of autopsies, the number of residencies, and whether or not there was an out-patient service. Beginning with the issue of 1937, a further valuable addition was made in that the posts were divided into residencies and assistant residencies.

A study of the figures submitted in these publications has been made, utilizing certain criteria for inclusion of a residency as desirable for men wishing to qualify for the American Board of Surgery. The criteria utilized in the selection were 20 per cent or more free beds* and an out-patient service. It was felt that if a hospital did not have at least this number of free beds, it could not possibly give to the young surgeon in his later years of training the *adequate operative experience in which the candidate had assumed the complete responsibility* as is demanded by the Board of Certification in Surgery. Utilizing these standards, we find the following number of approved residencies: 1934, 417; 1935, 439; 1936, 456; and 1937, 603. The figure for 1937 may be further divided into 248 residencies and 355 assistant residencies. These figures show a steady gain in the number of the posts available, but if one examines the figure critically it is noted that the great percentage of the posts offered are for one year only and there is no intimation given in the data that such a post may be continued a second year with increased responsibilities. Moreover, it is known that a very considerable number of these residencies are posts for which a man qualifies after a single year's previous training, training which, in fact, may be on a rotating service where the amount of surgical experience has been meager. It would be desirable if the Council on Medical Education and Hospitals could extend somewhat the amount of information given concerning the approved residencies and assistant residencies. Thus the addition of an item describing the amount of work the interne should have had before he can qualify for the post of assistant resident would be very helpful. There obviously is no comparison between the responsibility which can be given to a resident in surgery who has had a rotating interneship and the responsibility which can be given to a resident surgeon who has had not only a one- or two-year straight surgical interneship but in addition has had one or two years as assistant resident surgeon. There should be some way of bringing this information to the young surgeon. The splendid work accomplished by the Council of Medical Education and Hospitals in elevating the character and diminishing

*I have interpreted "free" or "charity" beds as beds occupied by patients who are a direct responsibility of the hospital rather than of a private physician and thus might be utilized as a source of material for giving experience to properly qualified men under the guidance of responsible members of the hospital staff. "Private" as opposed to "charity" patients obviously cannot be used for this purpose.

the number of our medical schools by a system of grading suggests that a similar plan be adopted in regard to hospitals.

In adopting a system of rating or grading hospitals for graduate training in surgery, it would be necessary to grade or rate them on a descriptive plan, grouping separately (1) hospitals approved for undergraduate teaching, (2) hospitals approved for graduate training, and (3) hospitals of great community value whose clientele is mostly private. Such a plan would place no affront on the splendid private hospitals performing a most useful community purpose, as would occur if only teaching or training institutions were graded. Moreover, such a grading of all hospitals would serve another most useful service by stimulating boards of trustees to better their own institutions. Thus, a Grade "A" Hospital in each group would be representative of the best qualifications in that group. In the group fitted for the graduate training of the surgeon, Grade "A" would signify hospitals which had some tie-up with a medical school in order that the qualifications under the head "Special Training" in the booklet of information put out by the American Board of Surgery be fulfilled. Moreover, in this group a Grade "A" Hospital would have to have some set-up whereby it could train a young surgeon for at least three years following his first year of interneship. Such restriction would unquestionably pull down the now 603 listed residencies and assistant residencies and would perhaps leave us in the Grade "A" group of those hospitals suitable for the graduate training of the surgeon a mere 10 per cent of the 603 residencies and assistant residencies listed in the 1937 educational number of the *Journal of the American Medical Association.*

If we consider that the country needs somewhere around 8,000 general surgeons, active, between the years of forty and sixty, and that in this group there is a 5 per cent mortality and a 5 per cent retirement rate yearly, then we need around 800 new surgeons each year. If all are to be qualified by the American Board of Surgery, it means that 800 suitable posts should be available each year in hospitals connected with medical schools. This does not mean that only hospitals in close physical relation with medical schools can be considered. It would be entirely feasible for those hospitals that are distant from a medical school to make some arrangements with a school whereby men at a certain point in their period of clinical training could receive more experience in the basic medical sciences in the medical school.

The task of increasing the number of posts where men can be adequately trained in surgery after graduation from medical school is the responsibility of each and every surgeon. We must all urge the establishment of further residencies in surgery and assist in every way to work out a liaison between medical schools and hospitals not now having such a connection. From the point of view of the care

of the patient, let alone this educational responsibility of the hospitals, there is every reason why adequate residencies should be established. The addition of such a person to a hospital staff leaves in the hospital at all times a man well equipped for surgical emergencies. It also leaves a mentor and leader for the house staff, someone who will see that the internes are taught the innumerable little things they should know about dressings and the care of the patient. Such an incumbent gives confidence to the patient and allows the trustees to feel that the hospital is adequately covered at times when the visiting staff are not at hand.

A great step forward was made when the American Board of Surgery set up such stringent qualifications. It now behooves all of us to make possible the postgraduate training of the young surgeon so that the qualifications demanded can be met and fulfilled.

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Is Judgment Necessarily Inarticulate?

THE beginner in medicine learns by a hard road certain elementary facts about the body and its abnormalities, facts chosen for their relationship to disease and the treatment of disease. When he first approaches the patient, he seeks to apply this knowledge to a particular clinical problem. At the outset his progress is slow and stumbling; his facts are incomplete and he has not yet learned their relative importance, nor the trick of combining them into a logical composite. As his experience with disease enlarges, these crudities diminish and, after a time, he becomes able to visualize the patient as a whole, the place of the presenting disease in the picture, and the rational treatment to be applied. When he has developed the ability so to apply facts tempered by experience that in a good proportion of instances the outcome of the case points to no mistakes in its handling, he is said to have developed judgment.

Essentially, judgment is a memory and association phenomenon, being developed more quickly the wider the professional reading and the more catholic the clinical contacts. The man who can most pertinently gather together scattered observations from his experience and make them refer to the individual case is the man most assured of exercising good judgment. But there is implied another factor, which we profess to recognize in the field of medical education; namely, the ability to express reasons for conclusions. If a doctor can say "I am proposing to carry out this treatment because . . ." and can clearly express the rationale of treatment, one is more impressed by his decision as embodying good judgment than if he is

able to say only "I am proposing to carry out this treatment but I cannot tell you why." We insist on the student's writing down his opinion and the reasons therefor. It is recognized as good hospital practice to demand that the surgeon before operation commit himself to a diagnosis and give the reasons for operating.

In spite of this accepted relationship between clear thinking (implying good judgment) and clear expression, there remains in the philosophy of the profession a conception that may be regarded as an anomaly; namely, the idea of the existence of a somewhat mysterious gift, which descends upon a man after a number of years of experience, called "Judgment," a faculty so esoteric that the reasons for its conclusions cannot be expressed in words. It needs no profound research in medical literature, particularly that of presidential, commencement, and memorial addresses, to gain the impression that this faculty is the more revered the less articulate it is. This conception even creeps into the clinical literature. One may quote, for example, from a recent paper in this Journal: "It is just here that the surgeon of experience can tell that the patient is much sicker than he appears to be. He will not be able to state why he thinks so, if pressed by his colleagues."

There is no doubt, of course, that articulateness varies between men of equal judgment; in some men thought readily is transmuted into speech, whereas in others the transmutation is difficult. That fact does not negative the principle, however, that the clearer the thought the more easily it is expressed. One need not insist that every act of judgment be analyzed in the spoken or written word. It seems desirable, however, to make the point that inarticulateness is not an essential quality of good judgment. One may wonder if the contrary idea may not represent a vestigial psychologic remnant of the medical beard and frock coat, or even of the priesthood age of medicine, when mystery was of the essence of practice.

The matter is of most importance and interest in relation to the growth of the young medical mind. The contradiction between the teaching that clear thinking means clear speech, and the conclusion that the acme of medical attainment is necessarily associated with inarticulateness, must be obvious to the beginner. The young doctor cannot be much censured if this contradiction should lead him to conclude that the former teaching is not of great weight when the best of his elders can reach their most brilliant conclusions without being able to tell why. The temptation is presented to neglect the intermediate steps that demand the hard discipline of putting reasons into words, and to wait inertly, but more or less hopefully, for the golden moment when the great gift of Judgment will descend like a halo.

Although few hold formal teaching positions, there is no established surgeon of this present day who does not teach; and there are few

who are not sensitive to the proper demands of the young assistant for instruction. It may not be presumptuous to suggest that in the daily contacts of hospital practice the surgeon make the attempt not only by precept but also, even more importantly, by example, to inculcate the idea that no essential mystery resides in the exercise of judgment, that implied or actual expression can be found of the reasons for any act of judgment, and that this hard way is the path toward the greatest reward in terms of further development.

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Recent Advances in Surgery

CONDUCTED BY ALFRED BLALOCK, M.D.

RECENT ADVANCES IN THE STUDY OF BURNS

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INTRODUCTION

THE study of burns has made rapid strides since the introduction of tannic acid by Davidson[1-5] from 1925 to 1927. These years may indeed be termed a turning point in the treatment of burns. Numerous variants of Davidson's original tanning technique have given rise to a multitude of new methods. Research and clinical observations have been made which support the tanning principle and, contrariwise, numerous opponents of this principle have put forth valid objections to it. Hence, at the end of ten years of use of the new treatments, somewhat of a perspective can be gained. They have, to a limited extent, been tested by time and it is appropriate to take stock of their efficacy.

Reviews of the treatment of burns before the tannic acid era are given by Sherman,[6] 1918, and by Ravdin and Ferguson,[7] 1925. More recent reviews are those of Seeger,[8] 1937; Bancroft,[9] 1937; Douglas,[10] 1934; Dunbar,[11] 1934; Barnes,[11a] 1933; Pack,[12] 1934; the book by Pack and Davis,[13] 1930; and the series of articles by Wilson,[14-20] 1928-37. Shorter reviews are those of Pack,[21] 1926; Snedecor,[22] 1935; and Pfohl,[23] 1936. Probably the best recent review of experimental work is that of Lambret and Driessens,[24] 1937. The most recent clinical review is that of Fasal,[25] 1937. Pack,[26] 1926, gives a discussion of the medicolegal aspects of burns and scalds. Fasal,[27] 1937, gives a discussion of the various causes of burns.

The number of deaths from burns during the last few years has averaged about 6,000 per annum in the United States. This about one-sixth the number dying from automobile accidents. However, since the latter represent a myriad of types of injury, the importance of study of burns becomes more manifest as with them, if treatment is efficacious, benefits may be obtained. The death rate from burns is improving of late[28] especially in younger children.[29] Riehl,[30] 1933, reports a very severe burn in a newborn infant with temporary recovery which he attributes to transmitted resistance.

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The method of Berkow,[31, 32] 1924, 1931, of estimating the surface area of various portions of the body in the study of burns is of great practical value, especially in any attempted statistical analysis of recovery percentage.

SHOCK IN BURNS

I. *Relation to Shock From Other Types of Trauma.*—Another influence that has affected the study of burns is that gained from the study of traumatic shock in general. Burns are but a type of trauma, thermal in this instance, and trauma is but a noxious influence which, when severe, overtaxes the compensatory and recuperative powers of the animal organism. Much work has been done on the subject of traumatic shock in the past ten years. Previously the chief theories as to the origin of shock were the nervous and the toxic theories. In 1930 a physical theory was introduced and more recently the adrenal gland has been implicated.

Important general studies on the subject of shock have been published by Cannon,[33] 1923, in book form and in articles by Cattell,[34, 35] 1920-1923; Cannon and Cattell,[36] 1922; Erlanger and Gasser,[37-39] 1919; Erlanger, Gesell, and Gasser,[40] 1919; Porter,[41] 1919; Rukstinat,[42] 1932; Orr,[43] 1935; Frazier,[44] 1935; Coonse, Foisie, Robertson, and Aufranc,[45] 1935; Andrews,[46] 1935; Phemister,[47] 1935; Blalock,[48] 1930; Parsons and Phemister,[49] 1930; Mann,[50, 51] 1915-1918; Mann and Essex,[52] 1935; Seeley, Essex, and Mann,[53] 1936; Lambret and Driessens,[54] 1937; Moon,[55-59] 1934-1936; and most recently, Rein,[60] 1937. It is to be remembered, however, as pointed out by Simonart,[61] 1930, that the problem of shock in burns may be quite separate from that of shock due to other types of trauma.

II. *Theories as to the Origin of Burn Shock.*—

A. *Nervous and Adrenaline Theory.*—A purely nervous theory of causation of shock and death in burns is not generally held. This is quite contrary to the case with traumatic shock in general where many workers support a purely nervous theory. In burns the immediate effect of the painful injury may be a syncope or primary type of shock, but usually by the time the physician sees the patient this is over. It is only later that secondary shock develops.

Consistent with the multiplicity of other theories of causation of shock in burns, several separate theories are attached to the adrenals alone. Both the medulla and cortex are indicted separately and besides several authors, arguing chiefly from necropsy evidence, have attributed death to the adrenals as a whole.

In this latter category Bardeen,[62] 1898, was one of the earliest experimenters. This author noted no pathologic changes in the adrenals in five necropsies. Weiskotten,[63, 64] 1917, 1919, on the other hand, found adrenal changes in ten necropsies following uncomplicated superficial

burns. Greenwald and Eliasberg,[65] 1926, and Berkow,[66] 1931, also found adrenal changes. Inouye,[67] 1933, and Lattes;[68] Salvioli;[69] Hartman, Rose, and Schmidt;[70] and Olbrycht[71] also reported work inculcating the adrenals. Brooks and Blalock,[72] 1934, however, present evidence to show that somewhat similar adrenal changes will follow hemorrhage and trauma to muscles, indicating that they are not specific to burns.

Freeman,[73] 1935, and Freeman, Shaw, and Snyder,[74] 1936, and Freeman,[75] 1937, have proposed a theory of shock that consists in essentially an overexcretion of adrenalin to keep the blood pressure near normal despite a lowered blood volume. If the blood volume decreases more, and this it tends to do because of capillary exudation with increased arteriolar resistance, the increased adrenal output can keep the blood pressure compensated only so long and then it collapses. Thus, this theory proposes that an overproduction of adrenalin is a factor. Saito,[76] 1932, believes, contrariwise, that in burns a deficiency of adrenalin is the chief factor and he has prolonged the lives of experimentally burned animals by intraperitoneal adrenaline injections (average length of life of injected burned animals sixty-three hours; of control burned animals, twenty-seven hours). Riehl,[77] 1928, on the other hand, does not believe that adrenalin is a factor.

Other writers have accused the cortex as being the site of injury with resultant hypofunction and death. Swingle, Pfiffner, Vars, Bott, and Parkins,[78] 1933; Swingle and Parkins,[79] 1935; and Donahue and Parkins,[80] 1935, reported experiments tending to show a similarity between adrenal cortical insufficiency and traumatic shock. The injection of "cortin" was said to control the insufficiency and combat the shock. This work was immediately supported by Heuer and Andrus,[81] 1934, and attacked by Freeman,[82] 1933, and Britton and Silvette,[83] 1933, amidst extensive polemics. Wilson, Rowley, and Gray,[84] 1936, have taken the practical application of extract of suprarenal cortex from this discussion. They used this extract in three cases of burns, two of which they believed would have died otherwise. However, they mention that no sweeping conclusions can be drawn from this small series.

B. *Toxic Theory.*—From the very start of interest in burns, the toxic theory has been in the forefront as an explanation for the cause of shock and resultant death. Scores of poisons have been indicted, most often on an entirely empirical or theoretical basis, and the very number of suspected toxic substances is one of the chief evidences against positive proof for a toxic basis. On the other hand, just because some of the recent work has been against a toxic theory for death in other kinds of shock, this has no absolute bearing on the subject of burns, for they represent a separate problem.

Parascandolo,[85] 1904, was one of the early experimental proponents of the toxic theory. In work on rabbits he reported that death was due to toxic poisoning caused by a circulating substance acting on the liver

and kidneys as well as other organs causing fatty degeneration. He also reported the blood of burned rabbits was toxic when injected into other animals. In an extensive monograph Pfeiffer,[86] 1905, in experiments on guinea pigs and rabbits reported that deaths in the first two to six hours are due to shock, while thereafter they are caused by decomposition products of burned protein which he reported he isolated.

Robertson and Boyd,[87, 88] 1923, reported very definite results in their search for a burn toxin. After producing skin, subcutaneous tissue, and muscle burns experimentally, they reported finding a burn toxin that was present in whole blood and cells, but not in the serum. Therefore it was absorbed on the cells. They reported that alcoholic extracts of burned skin would produce symptoms when injected intraperitoneally or intravenously into experimental animals, but that these results did not follow the injection of similar extracts of normal skin or skin of animals burned after death. They reported that blood from burned animals injected into guinea pigs produced toxic symptoms and "autopsy findings in the fatal cases were identical with those seen in burned animals." They claimed to have separated a thermostabile neurotoxic and a thermolabile necrotoxic element in their toxin, which toxin consisted of primary and secondary proteoses. Because of the presence of this toxin, they advised exsanguination transfusion and concluded: "From a purely clinical point of view, there seems to be no doubt that the burned tissues are responsible for the production of some toxin which is taken up by the blood stream."

Underhill and Kapsinow,[89] 1931, repeated the work of Robertson and Boyd on the injection of alcoholic extracts of burned skin and reported that the control extracts were fully as lethal as the extracts of burned skin and the lethal factor in either case was merely the alcohol. They correlated the irregular action of the control extracts with the lack of a statement in Robertson and Boyd's papers on the number of controls done. Underhill and Kapsinow concluded: "Our experience with burns leads us to doubt the existence of a 'burn toxin,' and to believe that the persistence of this viewpoint is an obstruction in the way of clarification of the burn problem."

Underhill, Kapsinow, and Fisk,[90] 1930, presented data which showed delayed absorption of such substances as strychnine and phenolsulphonephthalein from burned areas. Since there was fluid loss from the capillaries, they thought that these absorption experiments showed that the increased permeability was in one direction only, namely, from the blood into the tissues. Mason, Paxton, and Shoemaker,[91] 1936, performed experiments somewhat similar to the preceding ones, only they used a substance of low molecular weight for their absorption tests, sodium iodide. This was excreted in almost identical quantities in the urine of burned and control animals after subcutaneous injection, the

injection being made in the case of the burned animals into the skin of the burned area on the abdomen six hours after burning.

Harrison and Blalock,[92] 1932, performed experiments, the results of which opposed the toxic theory. These were repetitions of experiments that had been reported by others as favoring the toxic theory. (1) The transplantation of burned skin had no effect on the recipient animal. (2) The effects of débridement on the survival period of burned dogs: the survival averaged seven and one-half hours in the débrided series and seventeen and one-half hours in the control dogs. (3) Transfusion of blood from burned to normal animals was without effect. Blalock and his associates,[133-135] 1931, reported work favoring the loss of plasma-like fluid being a potent cause of death following burns (see section of physical theory), but Mason and others,[91] 1936, quote him as saying in 1934: "I believe that deaths which occur from three to ten days following severe burns are due in large part to the absorption of protein decomposition products." Straus,[93] 1937, working in Karsner's laboratory, was unable to isolate substances in burned guinea pigs which, when injected into other animals, would act like a toxin.

Japanese investigators, on the other hand, have practically invariably supported the toxic theory. Nagamitu,[94, 95] 1933, 1935, found that blood serum or fluid perfused through the burned hind limbs of dogs or rabbits when tested on cat's blood pressure or guinea pig's uterus or intestine showed a histamine-like depressor action. However, the action was not abolished by histaminase. Shimada,[96] 1934, believed that the burn toxin is nothing other than histotoxin which can be differentiated from histamine by its action on the toad's kidney. Miura,[97] 1934, perfused the burned hind legs of rabbits and found a toxin. Inouye,[67] 1933, has seen autopsies of three burned patients with changes in the adrenals. He believed the changes in burns are much the same as those caused by strychnine, barium chloride, and picrotoxin. He said that Pfeiffer,[86] 1905, attributed these changes in the adrenals (disappearance of lipoid bodies, loss of chromaffine affinity, hemorrhage, hyperemia, and focal necrosis) to the accompanying low blood pressure due to shock. He further quoted Kolisko, Nakata, and Niemeyer as postulating that a toxic action caused the adrenal changes and that the adrenal injury in turn killed the animal. On the other hand, Takeuchi said that the adrenal changes are only a part of the general picture and not the direct cause of death. Inouye, himself, adhered to Takeuchi's theory but believed also that the adrenal damage may be one of the factors in the causation of death. Ishizawa,[98] 1935, from experiments on rabbits, postulated that the hemolysis of red cells at the site of the burn gives rise to a toxic substance and is the cause of early death in burns. A hemoglobin discoloration of the plasma was often found as early as fifteen minutes after a burn. A study of this paper reveals that such a hemoglobin discoloration of the plasma was only observed at fifteen minutes except

in four animals where it was observed one hour after burning and that these latter animals recovered. The other animals, four of which died under three hours and the remaining four dying from ten to sixty hours after the burn, showed hemolysis only in the fifteen-minute sample. Furthermore, the hemolysis stopped while the blood was still concentrating, all of which tends to prevent one from accepting Ishizawa's postulates. Murai,[99] 1933, has studied the effects of injections of heated skin and gastric mucous membrane extracts, finding the former more toxic. Ravdin and Ferguson,[7] 1925, and Davidson,[1-5] 1925, 1927, favored the toxic theory. Davidson's whole tannic acid therapy was founded on the concept of prevention of toxin production by an absorption from injured or autolyzed tissue and was instituted at the suggestion of Mason.[91]

Harkins, Wilson, and Stewart,[100] 1935, presented work that was evidence against the toxic theory. They made protein-free extracts of normal and burned skin of rabbits by means of trichloracetic acid extraction. The extracts of normal skin contained a depressor substance which was not acetylcholine, adenosine, histamine, or the "P" substance of Euler and Gaddum,[101] 1931. Extracts of rabbits' skin, to which heat had been briefly applied at intervals of from three minutes to 48 hours previously, contained an apparently identical depressor substance. Such extracts showed no increased depressor activity, but sometimes a diminution, which is probably due to dilution of the depressor content of normal skin by edema fluid. Later, Wilson, Jeffrey, Roxburgh, and Stewart,[20] 1937, again used rabbits as the experimental animal, but this time they did not use protein-free extracts and this time the extracts did show evidence of toxic action. The edema fluid which accumulated in the burned area gradually acquired toxic properties and, when collected forty-eight hours after burning, it was frequently lethal to healthy animals of the same species. The development of toxicity was independent of the action of bacteria and seemed to be related to the autolysis of injured tissue. The toxic principles were associated mainly with the globulin fraction and their action was on the nervous and circulatory systems and in the production of degeneration of liver cells. This work is further discussed by Roxburgh,[102] 1936, and in an editorial in the *British Medical Journal*,[103] 1937.

Cattrano,[104*] Boyer,[105] Guinard,[106] Vegt and Vaccarezza,[107] Kozareff,[108] and Massabuau, Laux, and Ginestié,[109] 1935, favor the toxic theory. Bernhard,[110] 1936, found that extracts of experimentally burned skin were toxic when injected into other animals. From autopsy changes in the case of a girl aged four years dying seventeen days after a third degree scald of the left hand 9 by 5 cm. in area, with the observation of changes in the heart muscle and kidneys, Brenner,[111] 1936, favored the toxic theory. The size of this burn and death after the time when infec-

*Possibly an error in spelling Catiano.[112]

tion had taken place might indicate that death in Brenner's case was due to infection rather than autolytic toxins. It is to be pointed out that one definition of a burn is "an infected wound produced by heat." Catiano,[112] 1882, believed that when ammonium formate, derived from the sweat, was heated in the process of burning skin, it lost water and became HCN, the cause of the toxic action of burns.

Barsoum and Gaddum,[113] 1936, reported a rise in blood histamine in burned patients reaching a peak of five times normal seven days after the burn. They stated, however: "The relation of this rise in blood histamine to secondary shock is uncertain. There was no clear evidence of any correlation between the blood histamine and the clinical condition of the patient." The rise was possibly due to interference with the function of the kidneys or other histamine-excreting mechanisms. The rise occurred later than the time of secondary shock, but the writers attributed this to the fact that the patients were Egyptian!

Fender,[114] 1933, gave an excellent review of much of the earlier work favoring the toxic theory, listed seventeen toxins proposed by thirty-four different workers, and showed that many of their experiments are open to question (see Table I). Merely because there is confusion as to the name of the toxin is of course not evidence against its presence. Fender found changes in the spleen and lymph glands of burned rabbits, but he concluded: "The theory of causation of death by a toxin cannot be

TABLE I

PARTIAL LIST OF TOXINS POSTULATED AS THE CAUSE OF BURNS*

NO.	TOXIN	AUTHOR
1.	Ammonia	Edenbuizen
2.	Ammonia or urea	Billroth
3.	Fibrin ferment	Foa
4.	Urea	Ponfick
5.	Hydrocyanic acid	Catiano[112]
6.	Ptomaine	Lustgarten, Kitjanitzin, Parascandolo[85]
7.	Pyridine base	Reiss
8.	Hemolysins and hemoagglutinins	Von Dieterrichs
9.	Choline-like substance	Kohlrausch
10.	Methyl guanidine	Heyde
11.	Diamino acids	Eden and Herimann
12.	Primary and secondary proteoses	Robertson and Boyd[87, 88]
13.	Peptone	Olbrycht
14.	Inorganic intracellular material	Turck
15.	Unspecified protein cleavage products	Pfeiffer,[86] Fraenkel and Spiegler, Brancati, Il Seung O, Nishimura
16.	Unspecified toxin	Avdakoff, Boyer and Guinard, McCrae, Scholz, Vacarrezza, Bardeen,[62] Weiskotten,[63, 64] Pack, Davidson,[1-5] Speese and Bothe, Weidenfeld
17.	Potassium salts	Schjerning
18.	Toxalbumin	Dorrance and Bransfield
19.	Anaphylaxis	Tuder
20.	Abrin and ricin-like toxin	Stengel and Fox

*Adapted from Fender,[114] 1933, and Pack,[234] 1926.

said to rest upon adequate experimental work at present.'' Simonart,[61] 1930, also found no evidence for a toxic action. Lesser,[115] 1880, attributed the toxic action of burns to a functional impairment of the red cells that pass through the burned area making them foreign to the organism and giving rise to anemia in the functional sense.

A session of the 1937 French Surgical Congress (held in Paris in October) was devoted to the subject of burns.[116] Pierre Duval (cited also by Rudler,[117] 1935) gave a report favorable to the toxic theory. He believes that autolyzing tissue at the site of the burn acts as a toxin. He reported that in animals it was found that repeated burns conferred a state of sensitization, which in turn confers a certain immunity or resistance toward burns. Mourge-Molines advocated silver nitrate-tannic acid treatment to combat the toxemia and in the discussion McClure, of Detroit, spoke in behalf of tannic acid. Riehl, of Vienna, advocated blood transfusion, tannic acid, and, in many cases, saline baths. Wilson, of Edinburgh, advocated 20 per cent tannic acid, gentian violet, and acriflavine to combat infection and adrenal extracts to combat toxemia. Seemen, of Munich, advised electrocoagulation of the burned surfaces. Donati, of Milan, endorsed the methods which aimed to prevent dehydration and intoxication. Leriche, of Strasbourg, found that tannic acid treatment did not prevent humoral changes, and he uses mercurochrome in children and in infected burns. After the first few days of a burn, Piollet and Limousin, of Clermont-Ferrand, use cod liver oil dressings. The keynote of the entire meeting was in harmony with the toxic theory. Rudler,[117] 1935, was one of the first to use tannic acid in France.

The experiments of Rosenthal,[118, 119] 1937, are one of the most recent units in the evidence for the toxic theory. If these can be confirmed, they certainly represent strong reasons for adopting this idea. Rosenthal found a substance that caused contraction of the virgin guinea pig uterus in the blood of burned shoats, adult pigs [sic], guinea pigs, and human beings. This substance was at first linked with the red blood corpuscles, but it was later found in the serum. It differed from histamine in several ways. The sera of shoats, pigs, and human beings that were burned were tested in the healing stage and found to neutralize histamine and ''burn toxin'' as tested on the guinea pig uterus. However, normal serum has this action to some extent. No controls using blood concentrated by some other means than by burning alone were used in these experiments.

C. *Bacterial Theory.*—Aldrich,[120] 1933, at the suggestion of Firor, aroused interest in this theory and to combat bacterial action gentian violet was introduced as a method of treatment. For the first twelve hours, the burn areas were found to be sterile. After this period, it was found that in 100 per cent of the severely burned patients and in a large majority of the minor burns the beta-hemolytic or gamma strep-

tococcus could be grown from repeated cultures. The concentration of these organisms increased with the obvious signs of sepsis and the beginning of the toxicity of the patient, until after forty-eight to fifty-six hours pure cultures of the streptococcus could be obtained, having outgrown all the other organisms. Coincidentally, the characteristic bad effects of the burn were shown by the patient.

Clark and Cruickshank,[121] 1935, believed that infection of burns, particularly by *Streptococcus hemolyticus,* is common even with tannic acid and is responsible for the fever and toxemia during the first four or five days of the illness. Therefore, they added 20 per cent dettol to the tannic acid and even suggested the administration of scarlet fever antiserum. Cruickshank,[122] 1935, reported detailed bacteriologic studies of burns. He believed that the high incidence of infection by this organism is probably favored by the congregation of patients in wards, as he discovered hemolytic streptococci in the throats of patients more frequently during their first week in the hospital than at the time of their admission; these bacteria were numerous in the atmosphere and dust of the wards in which the burns were treated.

Marsh,[123] 1935, was an earnest and quite radical supporter of the bacterial theory, and, without showing in his paper that burns are affected by staphylococcal infection, went on to advise empirically that "every case of severe burn should receive intravenously at the earliest possible moment, an injection of staphylococcal antitoxin amounting, for an adult of normal size, to at least 5000 international units. . . . People constantly exposed to the risk of severe burns, by nature of their employment, could be treated prophylactically with a course of staphylococcal toxoid injections."

The writer,[124] 1936, reported that in a series of burns the only two fatal cases within the first three weeks resulted from sepsis. The first of these was a male, aged forty-four years, with second degree burns of the left leg, face, neck, thorax, and arms; third degree burns of hands and forearms, following a gasoline explosion. Blood concentration with hemoglobin, 140 per cent, and red count, 7,200,000, was noted twenty-four hours later. On the seventh day pyarthrosis of the left knee due to *Streptococcus hemolyticus* with a fever of 106.6° F. and death were noted. The second case, a child of four years of age, died eleven days after a second and third degree scald of the thighs, legs, buttocks, arms, one-half of the back, and almost the entire trunk anteriorly. The patient developed a peritonsillar abscess and thrombosis of the veins of the right arm with a fever over 104° F. each of the last six days of life and at necropsy a typical Curling's ulcer was found. These cases demonstrate that not only many of the toxic manifestations present after the first few days but also some of the complications of burns may be explained on the basis of a septicemia, although this is not conclusively proved. However, these cases do not have marked infection

during the first twenty-four to forty-eight hours, which is the time when shock with blood concentration and low blood pressure, low cardiac output, and low blood volume occur. Death from true secondary shock usually occurs in the first day after the burn, which is earlier than infection becomes active.

D. *Fluid Loss (Physical) Theory.*—Underhill and his associates,[125-132] 1923-1930, were the first to draw attention to the extensive local loss of fluid in burns. Previously (*vide infra*) the blood concentration resulting from such fluid loss had been noted. Underhill and his associates produced experimental burns and found that the edema at the site of the burn reached a maximum at the end of twenty-four hours and was reabsorbed in five to six days. Chemical analysis of this edema fluid showed it to be closely similar to blood plasma. Underhill measured the amount of this edema fluid by squeezing it out of the soggy tissues and found it to be equal in some instances to 70 per cent of the total blood volume.[131] Loss of such fluid is of much more serious consequence than loss of an equal amount of mere watery transudate. As the writer has pointed out, a person can urinate three liters a day, but can hardly part with a similar volume of blood in the same time.

Blalock,[133] 1931, determined the amount of fluid lost into the tissues by burning one side of the animal and then bisecting the animal and comparing the weight of the two sides. In a series of burned dogs the edema fluid averaged 3.34 per cent of the body weight (about one-half of the plasma volume). In plasmapheresis experiments Blalock found that 3.5 per cent of the body weight of plasma removed would kill the animals. The edema fluid in Blalock's experiments was similar to plasma as shown by Beard and Blalock,[134] 1931, and hence in the burned animals enough plasma was lost to kill them alone without invoking the action of any toxin at all, although the author makes no such sweeping conclusion. Johnson and Blalock,[135] 1931, next showed that the cardiac output was markedly decreased following burns.

The author,[136, 137] 1934-1935, performed experiments confirming Blalock's data when unilaterally burned animals were bisected. Furthermore he placed the burned animals on a tipping apparatus so that as the burned side grew heavier a kymographic tracing measured the weight shift. This furnished a graphic method of recording the local accumulation of fluid in experimental burns. The accumulation began at the time of the burn and continued in the form of a decelerating curve until death. Accompanying the collection of fluid was a simultaneous increase in the percentage of hemoglobin and in the hematocrit readings. After most of the fluid had accumulated, the fall in blood pressure set in and continued rapidly until death occurred in a state of secondary shock. The author,[138, 139] 1934, 1935, also showed that the bleeding volume of burned animals is markedly decreased. It is interesting in this connection that histamine does not appreciably lower the bleeding volume

under similar experimental conditions when it has lowered the blood pressure to a shock level. Schievers,[140] 1936, has shown that the blood volume is reduced by as much as 50 per cent in experimental burns. He attributed this partly to destruction of red cells but mainly to transudation of plasma. This work fits in with that of the writer on bleeding volume and along with that of Simonart,[141] 1928, is one of the bases for Schievers' support of the physical theory.

Lambret, Driessens, and Cornillot,[142, 143] 1937, and Lambret and Driessens,[54] 1937, also have shown that there is a marked blood concentration and blood volume decrease in experimental burns. These authors base their treatment of burn shock on replenishing the lost plasma.

Mc Iver,[144] 1933, presented some evidence that may be taken to support the physical theory. He showed that in human patients the blister fluid was similar to blood plasma and that the hemoglobin and hematocrit readings were markedly increased in such patients.

Gunn and Hillsman,[145] 1935, give credit to several theories in a graphic sequential representation of events following a burn. Mitchiner,[146] 1933, also gives some credence to the fluid theory.

Weiner, Rowlette, and Elman,[147] 1936, reported marked blood concentration with most red counts above six million, two being over eight million in forty burned patients. They pointed out that the low plasma protein, due in this instance to plasma loss, is quite different from that in dehydration from diarrhea or vomiting where the plasma protein is elevated, being in some instances over 10 gm. per 100 c.c. In this connection McClure,[148] 1936, pointed out that very little of the fluid lost in burns is due to evaporation, the slight increase being due to increased surface temperature.

III. *Treatment of Shock.*—The treatment of burn shock advocated by most workers is similar to that used to combat other types of shock. Fluids, warmth, stimulants, and sedatives are advised. Blood transfusion is especially advocated as by Riehl,[149, 150] 1932, 1933. Since the blood is concentrated in burns, the possibility of giving intravenous plasma rather than whole blood is to be considered. This procedure is not generally adopted, but it is used by Lambret and Driessens,[24] 1937, and by Weiner, Rowlette and Elman,[147] 1936. Wilson,[17] 1935, has used gum acacia.

Several writers have reported that large quantities of intravenous saline or Ringer's solution alone may be harmful in cases of traumatic shock. Davis,[151] 1937, reported such a finding in experimental animals. Beard and Blalock,[152] 1932, reported this to be true in intestinal trauma. If a crystalloid solution is injected, it carries more protein out of the blood stream than would have been lost were the animal left strictly alone. Blalock, Beard, and Thuss,[153] 1932; Beard, Wilson, Weinstein, and Blalock,[154] 1932; and Blalock and Beard,[155] 1932, have reported a similar finding in other types of traumatic shock. The observations of

these men might well be applied to burns to prevent possible harm to patients before the treatment of burn shock by injection of crystalloidal solutions is finally accepted. Certainly, until the matter is finally settled, if blood is available, blood should be given. Furthermore, even though the recipient's blood is concentrated already, the donor's blood is relatively dilute by comparison, so that the often cited objection to blood transfusion in the presence of blood concentration is not entirely valid. The use of saline and other crystalloid solutions should be undertaken with the full understanding that they may not only run right through the injured capillary walls but may carry valuable blood constituents with them, leaving the blood more concentrated in red cells and lower in plasma-volume than before. Working under other ideas, Gunn and Hillsman,[145] 1935, advise giving 5,000 c.c. of saline solution intravenously immediately in severe burns.

LOCAL TREATMENT OF BURNS

I. *Tannic acid.*—Chief credit for the introduction of tannic acid as a generally used therapy of burns should go to Davidson,[1-5] 1925-1927. Previous to this time tea had been used empirically by the Chinese (5,000 B.C.) and in the form of ink by the Jews and Filipinos. The older inks often contained more tannic and gallic acids than do the modern inks. McClure and Allen,[156] 1935, quote an article in the *Pittsburgh Medical Review*[157] of 1890 on the use of 5 per cent solution of tannic acid in burns.

The tannic acid treatment has been used extensively in this country by Fantus,[158] 1934; Penberthy,[159, 160] 1934-1935; Christopher,[161] 1930; Mason,[162] 1933; Bancroft and Rogers,[163, 164] 1926-1928; Beck and Powers,[165] 1926; Beekman,[166, 167] 1928-1929; Glover,[168] 1932; Walsh,[169] 1935; Seeger,[170, 171] 1932; Mc Elroy,[172] 1936; Wolfer,[173] 1936; Harkins,[124] 1936; Stanley-Brown,[175] 1935; and others. It has been used considerably in most of the European countries except France where Rudler,[117] 1935, is one of its supporters. Salzer,[176] 1928, of Vienna, reports good results following its use, while in Great Britain the treatment has been used especially by Wilson,[14-20] 1928-1937, and Mitchiner,[146] 1933.

Several aspects of the tannic acid treatment have been somewhat modified by recent reports. Seeger,[170] 1932, has shown the advisability of buffering the tannic acid solutions with sodium carbonate. Hartman and Schelling[174] also have studied the effect of pH on tannic acid. As done by Fantus at the Cook County Hospital in Chicago, buffering consists in using 7.95 gm. of anhydrous sodium carbonate to each 50 gm. of tannic acid that is used in preparing a 5 per cent solution. The use of a spray to apply the tannic acid instead of compresses as originally used by Davidson is now common practice except in the vicinity of the eyes. Various antiseptics have been added to the tannic acid to make it more bactericidal. Most authors believe that once pus has formed beneath

the tannic eschar it should be evacuated and the elevated eschar cut away. Martinez,[177] 1933, of Liverpool, stands alone in adhering to the theory of laudable pus and advising that the crusts with infection beneath not be removed.

Taylor,[178, 179] 1936, brought up an important objection to the use of tannic acid. This author showed by microscopic sections that in many cases of so-called third degree burns there are remnants of viable deep-lying hair follicles and sebaceous glands. These cells may provide a focus for new growth of healthy skin. Taylor further showed that these very cells may be killed by the tanning process and advised that the treatment be used only in cases of life and death. This is only one of the objections to tannic acid, others being that its eschar is stiff and either prevents motion around joints or, if allowing it, cracks; that it is not bactericidal to any great degree (see section below on gentian violet); that it may delay skin grafting (see section below on saline soaks); that it cannot be used around the eyes; and that its rigid eschar may cut off the circulation to the fingers, etc.

Despite these objections, it has a wide use in reducing the mortality of severe burns, whether it does this by reducing the loss of plasma or by preventing the absorption of toxic products. Table II (enlarged

TABLE II

REPORTED REDUCTION IN MORTALITY FOLLOWING INTRODUCTION OF TANNIC ACID THERAPY OF BURNS

AUTHOR	OTHER METHODS		TANNIC ACID	
	NUMBER OF PATIENTS	MORTALITY %	NUMBER OF PATIENTS	MORTALITY %
Bancroft and Rogers (N. Y.)	90	40.0	114	20.0
Beekman (N. Y.)	320	37.8	114	14.9
Wilson (Edinburgh)	300	38.7	117	11.1
Mason (Philadelphia)	91	28.5	87	13.3
Harris (Toronto)	–	26.6	–	12.0
Langer (Vienna)	86	16.3	65	7.7
Mitchiner (London)	243	9.4	249	2.4
McClure and Allen (Detroit)	118	9.3	358	11.7
Glover (Cleveland)	121	14.0	556	10.2
Hempel-Jorgensen	–	40.0	–	11.0

from Glover,[182] 1937) shows the reduction of mortality effected by the introduction of tannic acid in several clinics. It is to be remembered, however, that burns differ in degree and that with improving public health standards more smaller burns may be hospitalized than formerly. Also, the general hygienic care and the treatment of shock with blood transfusion, saline infusions, etc., may be more adequate than formerly. McClure and Allen,[156] 1935, reported no decrease in mortality. Mitchiner,[146] 1933, reported that the mortality may decrease from other reasons than tannic acid treatment, such as better hygienic care. He stated that in the London Hospital the mortality from 1899 to 1903 was 25.3

per cent and in another four-year period from 1924 to 1928 the mortality dropped to 6.6 per cent even though tannic acid was not yet introduced.

Most of the recently devised methods of burn therapy have been offshoots of the tannic acid precipitant method. Aside from the use of buffers mentioned above, considerable discussion has centered around the strength of solution to be used. Davidson originally introduced a 2.5 per cent solution; later most American clinics used 5 per cent, while in England, Wilson,[19] 1936, advocated a 20 per cent solution. Others have used various antiseptics with the tannic acid solution. Mitchiner,[180] 1933, advocated the use of 1-2000 mercuric chloride to make up the tannic acid solution. Wilson,[17] 1935, used 1-100 acriflavine and Clark and Cruickshank,[121] 1935, used 20 per cent dettol. Hartman and Schelling,[174] 1935, have studied the use of amyl tricresol in combination with tannic acid in vitro.

At the present time, as brought out in the discussion of Penberthy's recent article,[181] tannic acid spray is used at the Children's Hospital in Boston, tannic acid compresses at the Presbyterian Hospital in New York, and tannic acid baths at the Massachusetts General Hospital. Gentian violet is used at the Johns Hopkins Hospital and the New Haven Hospital. To these data it is to be added that tannic acid is used in St. Lukes Hospital, Chicago; Henry Ford Hospital, Detroit; Children's Hospital, Detroit; and in conjunction with silver nitrate at Children's Memorial Hospital, Chicago.

II. *Tannic Acid and Silver Nitrate.*—This modification of the tannic acid method was introduced by Bettman,[183] 1935, with the idea of hastening the tanning process. After application of 5 per cent tannic acid solution, "the entire area is sponged over with a 10 per cent solution of silver nitrate. The burned tissues become black almost immediately. All oozing and leaky areas are sealed at once and the patient is encased in an impervious antiseptic dressing that has formed in seconds instead of hours." Bettman reported on the further use of his method[184] in 1937; and it was adapted in New Zealand by Cotter and Kimbell,[185] 1935, and in England by Mitchiner,[186] 1936. In Sweden Kissmeyer,[187] 1936, used it in the form of an ointment on burns that are not severe enough to enter the hospital:

℞		
	$AgNO_3$	0.25
	H_2O	25.00
	Olive oil	25.00
	Wool fat	50.00

In a recent report Glover,[182] 1937, adapted the combined treatment instead of tannic acid alone such as he previously used.[168] With the silver nitrate, he now uses 10 per cent tannic acid. He states that with this combination coagulation is usually produced within thirty minutes and

that the speed of coagulation is very important from the standpoint of comfort and prevention of absorption.

III. *Saline Soaks.*—Blair and Brown,[188] 1931, and Blair, Brown, and Hamm,[189] 1932, have brought attention to the use of saline soaks rather than any chemical or precipitant treatment of burns. They point out that complete fixation retards the natural sloughing off of the dead tissues and thus delays the opportunity for surgical repair, and that caring for these wounds without resorting to tanning may require painstaking extra work, but it will be necessary for a shorter time and will win more worthwhile results. Plain water, physiologic solution of sodium chloride, or a mild antiseptic may be used, but a hypertonic salt solution seems to have special virtues. The soaking is usually done in a bathtub and the authors state that by its use most burned areas can be made sufficiently clean and the granulations sufficiently firm for grafting within from three to five weeks. Crile,[190] 1936, also uses the saline soak method. The excellent results from the standpoint of the eventual plastic result achieved by these men would suggest that it be used much more extensively and that it replace tannic acid in many instances. However, as the author[124] has pointed out, there are certainly some severe burns in which the immediate survival of the patient is of more imminent importance than the perfection of the ultimate plastic result, and in these instances, whether its action be by precipitating toxins or by preventing fluid loss, a tanning treatment does seem to reduce the immediate mortality. Furthermore, hypotonic soaks may be dangerous.

IV. *Other Methods of Burn Treatment.*—Some of these are modifications of the tanning method and others are not. The former will be considered first.

A. *Cutch Extract.*—Seeger,[191] 1935, advises a 5 per cent solution of Cutch extract which contains 60 per cent tannin and is less expensive than tannic acid. This author states in the same article that tannic acid (or Cutch extract) reduces pain, but "we have noted no reduction in mortality attributable to its use."

B. *Taktocut.*—The Jägers,[192-194] 1936, 1937, use taktocut, a somewhat similar tanning agent. In their hands this method has produced good healing without infection.

C. *Ferric Chloride.*—This method was used by Henry R. Slack,[195] 1915, of Lagrange, Ga., for a number of years, but it was never published by him. Slack found that when applying the tincture with applicators to the burned area, at first the alcohol caused more pain, but, as soon as the astringent effect occurred, the pain ceased. The application was repeated on return of the pain. The method was published independently by Coan,[196] in 1935 and now has some vogue. Coan uses the tincture or a 5 per cent solution.

D. *Gentian Violet and Other Antiseptics.*—These usually give an eschar that is more pliable than that produced by tannic acid and they

have the added advantage of being antiseptic in their action. These methods will be listed separately below.

1. *Gentian Violet (Methyl Rosanaline).*—This method was introduced by Aldrich,[120] 1933, at the suggestion of Firor. A 1 per cent solution is used as a spray in a manner identical to tannic acid. The method has since been used by De Hart,[197] 1936, and others. An objection to the gentian violet is that it discolors everything with which it comes in contact. Churchman and Herz,[198] 1913, showed that gentian violet was not very toxic. Certain rabbits were well, despite the fact that gentian violet in a concentration of 1-2500 was circulating in their blood stream. The dye disappeared from the blood promptly and stained the mucous membranes among other tissues.

2. *Brilliant Green.*—Koritkin-Nowikow,[199, 200] 1934, 1936, treated a series of 215 patients with a 1 per cent solution of brilliant green, 139 of which were second degree burns and 76 third degree. Of these nine, or 4.02 per cent, died.

3. *Brilliant Green Plus Acriviolet.*—Aldrich,[201] 1937, reports that this combination is preferable to gentian violet. He states that gentian violet is active in combating gram-positive organisms, e.g., *Streptococcus hemolyticus,* but that it is ineffective against the gram-negative organisms. The acriviolet used in the present method is a loose chemical combination between acriflavine and crystal violet and the new treatment provides the strongest antiseptic against the gram-positive organisms that will not injure living tissues and also has a high specificity against the gram-negative organisms. In broth cultures it is bacteriostatic in a concentration of 1-1 million against gram-positive organisms and in a concentration of 1-10,000 against gram-negative organisms. Referring to the gentian violet treatment itself, Aldrich states that in the two years following the introduction of this mode of burn therapy into the Johns Hopkins Hospital the mortality dropped from 42 to 13 per cent. Continuing with regard to the new dye combination, Aldrich says: "While the new dye is as superior to gentian violet as gentian violet is to tannic acid, I do not believe that it is the final answer to all the problems presented by a burned patient. I am certain, however, that the conception of a burn as an infected surgical lesion is correct, and that it is infection rather than absorption of a split protein which causes death in burns. For, let me add again, where there is no infection, there is no toxemia."

4. *Acriflavine.*—This substance is used by Robertson,[202] 1933, in a 1-800 solution and by Mummery,[203] 1933, in a 1-1000 emulsion in medicinal paraffine.

5. *Mercurochrome.*—This substance was used by Sorrel, Guichard, and Gigon,[204] 1935, and by Turner,[205] 1935. The latter author states that tannic acid is bad because: (a) the coagulum is not transparent so that pus can form beneath without being seen, resulting in destruction of

skin islands; (b) the skin bed is damaged; and (c) it is unstable. He finds that the use of a 2 per cent solution of mercurochrome overcomes these difficulties.

6. *Silver Nitrate Plus Gentian Violet.*—Just as tannic acid was supplemented by silver nitrate, so Branch,[206] 1935, uses the latter with gentian violet. In a series of eighteen cases he says that his method does away with several of the disadvantages of tannic acid, namely, too tough eschar, prevention of movement, lack of prevention of infection and killing of skin islands when the tough eschar is removed surgically.

7. *Dichloramin-T.*—This antiseptic has been in use longer than gentian violet and is usually administered in conjunction with paraffined gauze, such as by Lee,[207, 208] 1920, 1923, and by Ravdin and Ferguson,[7] 1925.

Methods independent of the tanning principle include:

A. *Cod Liver Oil.*—This method was introduced by Lohr,[209] 1934, and used by Holmes,[210] 1937; Steel,[211] 1935; Stevenson,[212] 1935; and Chevallier, Carcassonne, and Luccioni,[213] 1937. The cod liver oil is applied in the form of a wet dressing and is supposed to be mildly bacteriostatic and stimulating to the growth of new epithelium.

B. *Initial Cold Water Treatment.*—This method is used in Seattle by Rose,[214, 215] 1936, 1937, who has cut his mortality in half by its use. The patient is placed in a bath of water at 70° F. on admission and over a period of three hours this is heated gradually to 98° F., following which the patient is treated in the routine manner with either tannic acid-silver nitrate or ferric chloride. Greeley,[181] 1936, states that in scalds, if cold water is thrown on immediately, the scald may not penetrate so deeply. It is to be remembered, however, that the cold water treatment is in opposition to the generally recognized mode of dealing with shock in severely burned patients and aside from its temperature, its hypotonic effect on a raw oozing surface may be dangerous.

C. *Paraffined Gauze.*—Used by Sherman,[6] 1918; Paravacini,[216] 1928; Lee,[207, 208] 1920, 1923; and others.

D. *Viscopaste Bandage.*—Robb,[217] 1935.

E. *A mixture of dextrose, levulose, and sucrose* has been used by De Whalley,[218] 1935.

F. *Camphorated Oil.*—Mc Inturff,[219] 1936.

G. *Amyl Salicylate.*—Stewart,[220] 1937, has used amyl salicylate for minor burns where toxemia and shock are not factors. Since the substance has no bactericidal qualities, it is mixed with an antiseptic, such as "abricide" (a mixture of isomeric substituted phenols). Amyl salicylate can be used on infected burns and is preferable to other salicyl esters, although its bad smell is a definite disadvantage.

H. *Débridement.*—This method has been used by Ravdin and Ferguson,[7] 1925; Läwen,[221] 1936; and Arzt,[222] 1935. Opposed to this method are the ideas of those who believe that skin islands may be left (Tay-

lor,[178] 1936) and the experimental work of Harrison and Blalock,[92] 1932, who showed that burned animals are more likely to die if débridement is performed.

I. *Ultraviolet Light.*—Trusler,[223] 1935, believes that ultraviolet light prepares the granulations for skin grafting and uses the light as an adjunct to other treatment.

J. *Picric Acid.*—Because of its poisonous properties, this is not used in the treatment of extensive burns at present, being entirely supplanted by tannic acid. However, in the form of an ointment, such as butesin picrate, it can be used to advantage on small burns. Christopher,[224] 1928, advises its use in such cases.

K. *Other Methods.*—In a study of the method of treatment of 752 cases of burns, Willems and Kuhn,[225] 1936, listed forty-seven different methods. Of these, forty have not been listed elsewhere in the present paper. Since these burns occurred from 1932 to 1934, they represent a diversity of method of recent times. This very diversity may mean either that no one treatment is suitable to all cases, that the ideal method has not yet been found, or that the general medical practitioner is not yet informed of the best methods. A few of the various treatments listed by Willems and Kuhn include unguentine (44 cases), antipyrexol (35 cases), boric ointment (25 cases), picric acid (18 cases), zinc oxide ointment (18 cases), carofax (4 cases), onol (3 cases), ambrine (3 cases),* etc. It is quite possible that many of these were minor burns.

L. *Horse Serum.*—Introduced by Robinson,[227] 1917, and used more recently by Monteith and Clock,[228] 1929.

PATHOLOGY

I. *Local and General Changes.*—Inouye,[67] 1933; Fritz,[229] 1934; Dohrn,[230] 1901; Weimann,[231] 1927; and Schridde and Beekmann,[232] 1924, present data on pathologic findings. Bancroft's article,[9] 1937, in *Nelson's New Loose-Leaf Surgery* is a comprehensive review with illustrations. Hirota,[233] 1934, burned the hind legs of fifty-seven rabbits and made necropsy studies. He observed nuclear changes in the adrenals and alterations in the liver and kidneys. An extensive review of pathologic changes is given by Pack,[234] 1926, and some of the chemical aspects are discussed by Wells,[235] 1918.

II. *Blood Chemistry Changes Produced by Burns.*—According to the physical theory of production of shock in burns, the minor qualitative changes in the various blood constituents are of less importance than is the quantitative reduction in the amount of blood as a whole. This is said to be caused by a loss of plasma, and the fact that albumin may be

*The ambrine treatment introduced during the war is seldom used nowadays. It is mentioned by Willems and Kuhn,[225] 1936, as being used in three of their collected cases and is discussed by MacLeod,[226] 1918, in *Oxford Surgery*. Ambrine is a patented paraffin preparation.

lost in slightly greater quantities than globulin and sodium chloride to a larger extent than sugar depends merely on a somewhat selective permeability of the burned capillaries for smaller molecules. It is as though a city of a million population were literally decimated by an air raid, the average size and other characteristics of the populace remaining would be little altered in the aggregate compared to the great reduction in number of people. However, to carry the illustration further, it is true that warfare is often more lethal to adult males than others producing a qualitative alteration in the population as well.

Blood chemistry changes have been noted following burns, and to many they represent the cause rather than the result of the shock factors present. Davidson,[3, 5] 1926, 1927, made an extensive study of the chloride and protein metabolism in burns. He found a significant lowering of whole blood and plasma chlorides. This was believed due to a lowering of the plasma chlorides below the renal threshold rather than to a primary kidney change. Davidson also found a hypoproteinemia with especial reduction in the amount of albumin. Underhill and others,[128] 1930, also found a lowering of blood chloride (whole blood), while Mc Iver,[144] 1933, found normal plasma chlorides. It is to be pointed out in this connection that with such marked blood concentration and altered hematocrit and a difference in the chloride concentration of cells and plasma (vide Harkins and others,[236-238] 1929, 1931, 1932) a chloride study of both cells and plasma should be made in burned patients. Therefore, since the cells normally contain less chloride than plasma and since in burns the cells are increased, this would explain the finding of low whole blood chlorides on the basis of the hematocrit changes alone but would not account for the difference in the results on plasma chlorides reported by Davidson and Mc Iver. Baur and Boron's,[239] 1933, report of reduced low blood chlorides in five surviving men out of nine burned in an airplane accident is open to the same objection as is the work of many other writers.

Cicala,[240-243] 1935, working on rabbits, reported a decrease in the blood chlorides, and an increase in the nonprotein nitrogen. He believed that some of the increase in nitrogen is due to histaminelike substances. The creatinine and uric acid increased, the latter he believed being due to renal tubular damage secondary to traumatic shock. The reduced glutathione of the blood increased. Another Italian investigator, Titone,[244] 1936, also reported an increase of glutathione in the blood of experimentally burned animals. It is to be remembered that hematocrit changes alone may explain many of these changes in nonprotein nitrogen, etc., when done on whole blood. Christophe,[245] 1933, finds that the presence of albumin in the urine, increased nonprotein nitrogen, hypochloremia, and low A/G ratio become most marked ten days after experimental burns in dogs. Lambret, Driessens, and Malatray,[246] 1936, and Lambret, Driessens, and Warembourg,[247] 1936, have reported increased

nonprotein nitrogen and hypochloremia, but their results are not open to the criticism that blood concentration changes alone may account for their alterations as their protein analyses were done on plasma and cells with reduction in both instances. Douglas,[10] 1934, cites blood chemical changes following burns.

Mattina,[248] 1935, reports an increase in the concentration of the blood magnesium of six rabbits burned by thermocautery, boiling water, or paraffin. The increase began twenty-four hours following the burn, reached its maximum in ten days and returned to normal in forty days. Fazekas and Bacsich,[249] 1934, report an increase in lipoid bodies in the blood leucocytes beginning in five hours, reaching a maximum in twenty-four to twenty-eight hours, and returning to normal after five to six days or longer. In fatal cases these bodies increase up to twenty-four hours and an hour before death may suddenly drop to below normal in number. Slocum and Lightbody,[250] 1931, reported that in both normal and adrenalectomized rabbits the sugar and lactic acid concentration of the blood is increased following burns. Since the adrenalectomized rabbits also showed the rise, this is interpreted as indicating that adrenal hyperfunction is not a factor. Van der Hulst,[251] 1937, reported a decrease in the rapidity of sedimentation of red cells during the first twenty-four hours with marked increase thereafter. These changes were not due to infection or blood concentration. Fasal,[252] 1937, found a shift to the left and toxic granules in the leucocytes.

Analyses of the edema fluid that leaks out into the tissues after burns are of interest. The chloride content is a little higher than in plasma, the sugar and nonprotein nitrogen contents are about the same, and the protein content about 80 per cent of that of plasma (Beard and Blalock,[134] 1931; Underhill and Fisk,[127] 1930).

III. *Blood Concentration.*—One of the chief blood changes following burns is a blood concentration occurring soon after the injury and subsiding in a few days in cases with recovery. Following this an anemia develops due to the combined effects of loss of blood from the granulating area, poor nutrition, and internal destruction of blood. Early writers to report blood concentration included Baradue,[253] 1862; Tappeiner,[254] 1881; and Hock,[255] 1893. Hemoconcentration was reported by Locke,[256] 1905. Of ten cases of burns at the Massachusetts General Hospital, Locke found a red count over 9,000,000 in four of them. Pfeiffer,[86] 1905, also noted hemoconcentration as did Underhill, Carrington, Kapsinow, and Pack,[130] 1923. These latter authors studied twenty-one persons burned in a New Haven theater fire with a hemoglobin from 117 to 209. Mc Iver,[144] 1933; Shimada,[96] 1934; Lambret, Driessens, and Cornillot,[142] 1937; Lambret and Driessens,[24] 1937; Weiner, Rowlette, and Elman,[147] 1936; Douglas,[10] 1934; all noted hemoconcentration in burned patients. Simonart,[141] 1928; Blalock and his associates,[133] 1931; and Harkins,[124, 136-139] 1934, 1936; have also noted hemoconcentration in experi-

mental burns. Ravdin and Ferguson,[7] 1925, noted a hemoglobin of over 100 per cent in all but two of their clinical cases and the red cell count was proportionately increased. As previously mentioned, hemoconcentration also accompanies shock from other causes.

IV. *Curling's Ulcer.*—This complication of burns was described by Long,[257] in 1840, and by Curling,[258] in 1842. Since then sporadic reports of peptic ulcer following burns have appeared in the literature including recently the reports of Willems and Kuhn,[225] 1936; Wilson,[17] 1935; Glover,[182] 1937; Torchiana,[259] 1936; and Kapsinow,[260] 1934. The last named author attributes them to blood stasis and concentration with capillary oozing and rupture, while other authors believe them to be toxic in origin. Glover,[182] 1937, states that bleeding from a Curling's ulcer accounted for 4 per cent of his deaths. Four other patients developed ulcer without death, three being diagnosed by x-ray and one ulcer requiring late gastric resection. The author does not state how many of these ulcers might have existed before. However, the two deaths were in children and this practically precludes a preexisting ulcer.

Underhill, Kapsinow, and Fisk,[125] 1930, showed experimentally that in the production of a burn heat may penetrate the interior of the body to a degree sufficient to increase temporarily the temperature of the body cavities by several degrees. They regarded such an increase of temperature as sufficient to lead to local circulatory changes inducing the formation of ulcers, hemorrhages, etc., which from time to time have been ascribed to the effect of a burn toxin. It should be pointed out in this connection, however, that clinical cases of Curling's ulcer have been reported in which the abdomen was not burned. The only case of Curling's ulcer in the writer's series,[124] 1936, occurred in a patient with marked sepsis: a peritonsillar abscess, fever of 104° F. or more for six days before death, and thrombophlebitis of the veins of one arm. In such a case bacterial emboli are to be considered in the etiology.

V. *Miscellaneous Complications.*—Pack,[261] 1926, summarizes burns in various locations in this connection by saying: "Burns of the scalp are peculiarly susceptible to erysipelatous inflammation, but are not so often followed by cerebral mischief as one would think. When the burn is limited to the extremities or to the back, where the thick dorsal muscles serve as effectual protection to the subjacent viscera, the outcome is encouraging. However, burns of the abdomen, with the dangerous visceral proximity, have the highest mortality. Burns of the genitalia, the anterior thoracic surface and the face (over the area of trigeminal distribution) cause symptoms and dangers far out of proportion to their area allotment. Mucous membrane involvement, especially of the pharynx and larynx, adds to the gravity of the case. Burns of the flexor surfaces are more serious than on the extensor surface." Pack further

states that convulsions are practically always fatal and albuminuria and persistent vomiting are ominous signs.

Tetanus has been frequently reported as a complication of burns. Newberger,[262] 1912, reported 2 personal cases and collected 47 from the literature. Of these 49 cases, 38 died, making a 77 per cent mortality. Fasal,[263] 1935, reported on the extensive burn material in the skin clinic of Vienna. Of 2,327 burn cases seen from 1905 to 1930, not a single one developed tetanus, but in both 1932 and 1934 there was one case and thus he believes prophylaxis is to be considered. Kudrinsky,[264] 1936, gave case reports of tetanus following burns. Willems and Kuhn,[225] 1936, stated that of 752 collected burn cases only 23 received antitetanic serum, but as none of the other 729 cases developed tetanus, the physicians in attendance on those particular cases evidently did not use poor clinical judgment in refraining from administering serum.

In many clinics, including that of the writer, it is the practice to leave the right lower quadrant free when taking skin grafts in burn cases. This is considered especially advisable in children in whom the appendix has not been removed. Rose,[215] 1937, reported a case in which appendectomy was performed through a ferric chloride eschar twenty-three hours after a burn of the abdomen and thighs. The wound healed without drainage and six weeks later that area was skin grafted along with the surrounding granulating region and the patient recovered.

Fat embolism is a rare complication of burns but Strassmann,[265] 1933, reported on 125 cases of fat embolism due to blunt force and 5 following burns. He quotes Carrara as saying that in 46 per cent of burn cases coming to autopsy fat emboli are found.

Globus and Bender,[266] 1936, reported an extensive study of severe burns in an eight-year-old boy leading to fatal disseminated toxic degenerative encephalopathy (disseminated sclerosing demyelination). Clavelin and Hugonot,[267] 1936, reported a case with late formation of edema. They attribute the edema to low plasma proteins resulting from albumin loss from the granulating surface. Froment, Thiers, and Brun,[268] 1936, reported a case of burns with resultant anuria, hypertension, and bradycardia. However, their patient was burned so slightly that the sequence of cause and effect is not definite. Massabuau, Laux, and Ginestié[109] 1935, reported a burn with jaundice occurring on the third day.

It is quite possible, as was discussed in the section on the bacterial theory of death following burns, that many of the complications of these injuries are secondary to sepsis. Pfaunder and Schlossmann,[269] 1935, mentioned several complications, e.g., hemoglobinuria. They quoted Kruse who reported encephalitis with acute hydrocephalus and amaurosis in a fifteen-month-old child the day following a second degree burn. Scarlet fever or typical scarlet-like exanthema with rise in temperature may appear after severe burns. Some (such as Leiner) are wont to

regard these all as true scarlet fever, since quite frequently an angina, raspberry tongue, and desquamation and even transmissions occur. The possibility of a toxic erythema due to resorption of poisonous products from the damaged skin is to be kept in mind.

VI. *Malignancy Developing as a Late Result of Burns.*—Treves and Pack,[270] 1930, have reported a large series of carcinomas developing in burn scars (Marjolin's ulcer) treated at the Memorial Hospital in New York. Of 1,091 squamous cell and 1,374 basal celled carcinomas treated during the twelve-year period, 1917 to 1929, 21 (2 per cent) of the former and 7 (0.3 per cent) of the latter followed burns. The average age of the patients as a whole was about fifty-nine years and was fifty-four years for the burn cases. However, the age of the scar is more important than the age of the patient, as one case burned at the age of three years developed carcinoma at the age of seventeen years. It is of interest that of the entire 28 patients with carcinoma following burns, not 1 was ever treated by skin grafting. In 9 patients with a known healing time, 3 never healed, and for the other 6 the average healing time was 805 days. The average age of the scar was 32.5 years. Stauffer,[271] 1928-1929, reported a man with skin carcinoma developing on the face thirty days after a burn. As he had precancerous lesions elsewhere, the burn may have been coincidental. Johnson,[272] 1926, reported 4 cases of carcinoma seen at the Steiner Clinic in one year. Three of these cases were four years, eight years, and two years respectively in healing. The fourth case never healed and wore a dressing for sixty-one years. At that time (previous to Treves and Pack's report), Johnson could find no record in the literature of basal celled carcinoma developing in burn scars. Harkins,[124] 1936, has recently reported 2 cases of sarcoma following burns.

PLASTIC AND LATE TREATMENT OF BURN ULCERS AND SCARS

A trend towards the adoption of the thick Thiersch or "thick split" graft advocated by Brown and Blair has been noted in recent reports,[188, 189, 273-277] 1929-1936. Many other writers favor the use of the Reverdin, "pinch," or small deep graft of Davis,[278-284] 1929-1934, and this graft is still used in many instances, especially for children,[124] 1936, or over extensor surfaces. Homogenous grafts,[285] 1937, are sometimes used as a temporary life-saving measure by Bettman,[286] 1938, the idea being that even though these grafts do not take they will act as a protective covering for a few days or weeks. Bettman says in this regard that there is general recognition of the fact that homogenous grafts do not take permanently. However, during the three to five weeks that the grafts are adherent, the covered area is cleared of infection and the patient is saved the disastrous effects of septic absorption from those areas. By the time the grafts come away, the patient's condition is so greatly improved that measures which were not thought of earlier may

now be carried out with safety. Whole thickness grafts have been used in the repair of scars to advantage by Padgett,[287] 1937, while Ashley,[288] 1937, has used discarded foreskins preserved in ice cubes. Goode,[289] 1935, takes the thick split graft of Brown and Blair and cuts it in pieces after placing it epidermal side down on gutta percha. These pieces, about 2 cm. square, are then applied, with the gutta percha still attached, to the burned ulcer in the same manner as are pinch grafts. This author treats the donor area with tannic acid and fastens large thick split grafts to the recipient area by plastering the margins with collodion.

The later treatment of burn scars and contractures by pedicle grafts and other plastic methods has been described in several recent publications,[290-301] 1924-1937.

RELATED THERMAL AND ACTINIC TRAUMA

I. *Freezing.*—Harkins and Harmon,[302-306] 1934, 1935, 1936, 1937, have shown that many of the changes following freezing are similar to those following burns. A marked exudation of plasma-like fluid into the local tissues occurs with thawing, resulting in blood concentration, lowered bleeding volume and decrease in arterial pressure. This condition may be so severe as to be related to death of experimental animals. In these experimental animals a marked prolonged hypothermia was noted of about the same degree as that reported by Reincke,[307] 1875, in human beings (24.0° C., 75.2° F.).

II. *Sunburn.*—This usually produces a first or even second degree burn over the entire exposed surface area, involving especially the extensor surfaces, and it may be serious in some instances.

III. *Roentgen and Electrical Burns.*—Davis,[308-309] 1933, has made an extensive study of these burns. He concludes that early and wide excision of deep burns caused by Roentgen rays or radium, with closure of the defect thus made by tissue shifting, offers the best method for elimination of pain, prospects for permanent healing, and safety from subsequent malignant degeneration.

Electrical burns are noted for their depth, sharing this characteristic with Roentgen burns. They may erode large vessels and cause delayed death by secondary hemorrhage.

COMMENT

From the mass of work published in the past ten years, much of which shows no uniformity of opinion, it is difficult to arrive at arbitrary conclusions. In review, however, it seems that after a severe burn a patient may be subject to some degree of psychic or primary shock which usually quickly passes away. Following this, from the second to about the forty-eighth hour, secondary shock may ensue with marked blood concentration. Accompanying this is a moderate alteration in some of the chemi-

cal constituents of the blood and a marked exudation of plasma-like fluid into the burned area and from the burned surface, with, in experimental animals, lowered blood volume, bleeding volume, and cardiac output. Whether this stage of shock with resultant lowered blood pressure is due to these physical changes or to the production of a toxin, there is no uniformity of opinion.

Irrespective of whether this secondary stage is due to the physical effects of local fluid loss or to the poisonous effects of toxin absorption, it seems advisable in serious cases to administer an eschar-forming type of treatment to the local lesion. Such local treatment will seal off the tissues and prevent fluid loss or toxin absorption. Along with the local treatment, the usual supportive measures against shock should be used. The use of adrenal cortical extracts and blood plasma transfusions are to be considered. The choice of local treatment lies between numerous tanning agents. If the ultimate plastic result is of prime importance and the burn is not severe enough to endanger the life of the patient, escharotics may be omitted and saline baths used instead. If one adheres to the bacterial theory of toxin production, an antiseptic may be combined with the escharotic or used as a mild escharotic.

The presence of infection, usually *Streptococcus hemolyticus,* is a factor in the morbidity in the third stage of burns from the second or third day on during the next few weeks, especially the first ten days. At this time, if the patient has recovered, fluid loss is no longer an important factor except in producing a type of nutritional edema. The blood now becomes dilute and transfusions are necessary to overcome anemia rather than to combat shock. Toxins may or may not play a rôle at this time.

In the fourth or healing stage of burns, early skin grafting is advisable and in this work the use of thick split grafts is favored in many of the most recent reports.

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Related Thermal and Actinic Trauma

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Roentgen and Electrical Burns

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Review of Recent Meetings

REPORT OF THE FORTY-SEVENTH ANNUAL MEETING OF THE WESTERN SURGICAL ASSOCIATION, INDIANAPOLIS, IND., DEC. 3 AND 4, 1937

WARREN H. COLE, M.D., CHICAGO, ILL.

(From the Department of Surgery, University of Illinois College of Medicine)

DR. **LOUIS RASSIEUR,** St. Louis, Mo., **A Clinical Study of Congenital and Familial Hemolytic Icterus:** In a family consisting of mother, father, and three children, all except the mother suffered from familial hemolytic icterus. Because of jaundice, anemia, etc., the father had a splenectomy three years before he was married. He died at the age of thirty-seven years with uremic coma. All three children were girls. In the first child anemia, slight jaundice, and weakness appeared early. Examination of stomach contents revealed no free hydrochloric acid. Splenectomy was performed. Gallstones were present in the gallbladder. The second child presented similar manifestations, including stones in the gallbladder and absence of hydrochloric acid in the stomach contents. Platelets numbered only 28,000. Manifestations of the third child were similar to those of the other two. Platelets numbered 26,000, but they rose to 600,000 after splenectomy. Splenectomy was performed on all three children with relief of symptoms, but slight icterus persisted. In discussion **Dr. H. H. Davis,** of Omaha, Neb., called attention to the frequency of hemoclastic crisis following transfusion before splenectomy in patients with familial icterus. **Dr. G. M. Curtis,** of Columbus, Ohio, called attention to the fact that the disease is genetic, i.e., carried by the chromosomes.

Dr. Charles B. Puestow, Chicago, Ill., **Choledochostomy with Studies of Intraductal Pressure:** The essayist called attention to errors in diagnosis as one of the most common causes of poor results following cholecystectomy. Studies on intraductal pressure within the common duct in animals and human beings revealed a lowered pressure following cholecystectomy. This lowered pressure suggests that there was a reflex disturbance of the denervation of the sphincter of Oddi caused by the procedure of cholecystectomy. This finding of the lowered pressure within the duct was encountered in all of the patients reported in this series, upon whom cholecystectomy had been performed and the common duct drained. The hypothesis is suggested that the relief of symptoms following cholecystectomy may be due, in great part, to this relaxation following cholecystectomy. In a series of patients examined at autopsy the circumference of the common duct was larger in those upon whom a cholecystectomy had been performed. This finding has been reported by other authors, although it is not entirely consistent in animals. As yet there is no satisfactory explanation available to explain this dilatation of the common duct.

Dr. R. Russell Best, Omaha, Neb., **Cholangiography Demonstration of the Remaining Common Duct Stone and Its Nonoperative Management:** The fact that over one-third of the patients having gallstones in the common duct are not jaundiced is a factor which makes exploration of the common duct so important,

particularly when certain indications exist. A few patients were reported in whom the common duct was drained with a T-tube, but in whom all stones were not recovered from the common duct at the time of operation. In such cases an attempt should be made to encourage the passage of the stones in the common duct without another operation. A routine consisting of the administration of atropine and nitroglycerin, and the injection of warm olive oil and saline solution through the T-tube has been developed in order to encourage the passage of the stones through the sphincter of Oddi. If the stones found in the common duct after operation are proximal to the T-tube, the tube will obviously have to be removed before the stones can pass through the duct. On one or two occasions when the routine mentioned above was ineffective in obtaining passage of the stone, the common duct was irrigated with ether in an attempt to dislodge or break up the stone. In the case in which this method was attempted, the procedure was not effective, however. Cholangiography at the time of operation is advised to aid the surgeon in finding stones in the common duct.

Dr. Charles W. Mayo, Rochester, Minn., **A New Method of Repair of Complete Rectal Prolapse:** Attention is called to the fact that many cases of severe prolapse of the rectum are associated with a hernia of the median perineal type, and in which small intestine may herniate into the prolapsed rectum. In dealing with this type of complete prolapse, a low midline incision is made and the neck of the sac, through which the small intestine herniates into the rectum, is brought upward. The floor of the pelvis, which is very lax, is reconstructed and reinforced with fascia lata removed from the thigh. Care is taken not to apply sutures in the bladder wall. The sigmoid is anchored somewhat by suturing epiploic tags to stable structures.

Dr. John Alexander, Ann Arbor, Mich., **The Indications, Technique, and Results of Surgery for Bronchiectasis** (In the absence of the guest speaker, Dr. Alexander, this paper was presented by **Dr. Cameron Haight,** Ann Arbor, Mich.): The institution of conservative therapy is emphasized before the radical procedure of lobectomy is performed. Conscientious postural drainage, pneumothorax, and phrenic interruption were discussed as procedures to be used before lobectomy, but unfortunately such measures are rarely curative. Occasionally patients are made worse by phrenic interruption. Bronchoscopy is of great value in determining the presence of bronchial stenosis as an etiologic factor in the development or presence of bronchiectasis. The essayist has practically discarded thoracoplasty in the treatment of bronchiectasis. Lobectomy is indicated, particularly in the unilobar disease. The results of bilateral lobectomy are obviously not good; poor results have discouraged the author from its continuation. In bilateral bronchiectasis it is usually preferable to perform lobectomy on the bad side and to wait considerable time to see if conservative treatment of the less diseased lobe will not be effectual. Prophylactic lobectomy in mild bronchiectasis is not favored, chiefly because the mortality following lobectomy is not insignificant. The x-ray, utilizing the injection of iodized oil, is very useful. One lobe should be injected, and lateral as well as oblique views should be taken. The author favors the two-stage operation and calls attention to the lowering of mortality from 8 to 14 per cent down to 4 per cent by the delayed two-stage operation of Churchill. Eleven total pneumonectomies were performed in the author's clinic for disseminated bronchiectasis with five deaths.

Dr. Casper F. Hegner, Denver, Colo., **Some Points in Surgical Collapse for Pulmonary Tuberculosis:** Conservative measures, including pneumothorax, phrenic interruption, and scaleniotomy, should be considered before thoracoplasty is advised. The author is of the opinion that temporary phrenic interruption produced by crushing of the nerve should be utilized more often than it is. In discussion

Dr. Joseph W. Gale, Madison, Wis., called attention to the effectiveness of extrafascial apicolysis of Semb. In this operation he anchors the intercostal bundles to the first intact rib. Portions of the first three ribs are usually removed.

Dr. William A. Taylor, Ellensburg, Wash., **Congenital Cyst of the Lung:** Although congenital cysts of the lung are not common, they offer an extreme amount of difficulty in therapy, particularly if they become infected. They do not respond well to drainage. The type of surgical approach varies depending upon the type of cyst present. Although excision of the cyst may be possible, frequently pneumonectomy will have to be performed because the cysts are so often multiple. In the case reported the author was able to excise the cyst successfully.

Dr. Stuart W. Harrington, Rochester, Minn., **Pharyngoesophageal Diverticulum:** The term pharyngoesophageal diverticulum is a poor one since the condition is in reality a herniation of the pharynx. These diverticula usually are present on the left side. They are apparently of congenital origin. The frequency of esophageal diverticulum makes esophagoscopy advisable in all patients with dysphagia. In the series of 33 cases reported, the operation was performed in one or two stages. In the latter type of operation, the first stage consists of immobilization of the diverticulum through a long incision in the neck usually on the left side. The second stage is performed seven to thirteen days later. This adds to the safety of the procedure because the area is walled off by the granulation produced by the first operation. Feeding for several days is restricted to that effected by means of a nasal tube. In 25 patients upon whom a two-stage operation was performed, 6 had a postoperative fistula, 2 had a recurrence, and 21 had relief from symptoms. In 8 patients upon whom the one-stage operation was performed, no complications were encountered and all were relieved of symptoms. The point was made, however, that these statistics should not condemn the two-stage operation.

Dr. Edgar L. Gilcreest, San Francisco, Calif., **Lesions of the Shoulder:** The author emphasized the fact that most of the patients complaining of pain in the shoulder in the absence of fracture suffered from extra-articular lesions and not from arthritis. Lesions consisting of fraying or rupture of the supraspinatus tendon are not uncommon. A great deal of the symptoms referable to the shoulder are related to the fact that the shoulder joint is now a weight-carrying joint rather than a weight-bearing joint. The grating or crepitus which can be demonstrated so commonly in patients complaining of pain is rarely due to arthritis, but usually due to fraying and roughening of the tendons. Lesions of the biceps and triceps tendons were included in this discussion.

Dr. Kellogg Speed, Chicago, Ill., **Spondylolisthesis:** Displacement of a vertebra as encountered in spondylolisthesis may occur at any level, but usually it occurs at the level of the fifth lumbar vertebra and sacrum. The lesion is frequently traumatic, but it may be associated with incomplete fusion of the neural arch. The more conservative methods of treatment, including the use of braces, corsets, traction, etc., were discussed. Operative procedures consist primarily of attempts to obtain bony fusion by an anterior or posterior operation. The anterior approach is usually easier than the posterior. The procedure described by the author is to drill a hole through the fifth lumbar vertebra into the sacrum, and to drive a bone transplant obtained from the tibia into this opening. This transplant affords fixation and is effective in the treatment.

Dr. J. Dewey Bisgard, Omaha, Neb., **Fractures Involving the Epiphyseal Cartilage:** In a résumé of 232 fractures the author noted that significant injury

to an epiphysis was inflicted in slightly over 20 per cent of the cases. Injury to the epiphysis is very apt to result in prevention of normal growth, even though reduction may be adequate. Occasionally growth is interfered with only on one side of the epiphysis, thereby producing a deviation type of deformity, toward the injured side. Such deformities do not manifest themselves for several months after injury. In about one-half of the cases suffering from epiphyseal injury the x-ray will reveal disturbance in growth in about half the cases.

Dr. Eslie Asbury, Cincinnati, Ohio, **Internal Fixation of Hip Fractures Without an Open Operation:** Attempt has been made to treat fractures of the hip by internal fixation without resorting to such radical procedure as the use of the Smith-Petersen nail. The method used by the author consists of the introduction of three or four Kirschner wires through the trochanter into the head of the femur. Patients treated by this method should not be allowed to bear weight on the extremity under four or five months. When fractures of the femur are treated by Kirschner wires, attempt should be made to obtain slight over-correction of the deformity. In 39 cases reviewed, 28 had union.

Dr. James R. McVay, Kansas City, Mo., **The Treatment of Diverticula of the Bladder:** The details of preoperative and postoperative treatment of diverticula of the bladder were presented. The diverticula were dissected out and the cut edges brought together with No. 00 chromic catgut. The bladder is kept empty with a retention catheter which is attached to a suction apparatus similar to Wangensteen suction. In discussion, **Dr. H. L. Kretschmer,** Chicago, Ill., remarked that many diverticula need not be resected if the etiologic factor, which is usually an obstruction, can be removed.

Dr. Henry McGraw, Denver, Colo., **Intestinal Obstruction:** The author traced the development of the various procedures advised from time to time as supplementary aids in the treatment of intestinal obstruction. The use of Wangensteen suction has resulted in a lowering of mortality and the elimination of operation in the treatment of many cases. One of the dangers in the use of Wangensteen suction is that the surgeon may misinterpret the slight improvement which always follows the institution of Wangensteen drainage. If operation is necessary and is delayed, the mortality will obviously be increased. In discussion, **Dr. Thomas G. Orr,** Kansas City, Kan., called attention to the fact that enterostomy is seldom used any more in the treatment of intestinal obstruction since it is not needed when drainage through the opening is effected and since it is of no value when no drainage is effected. **Dr. Urban Maes,** New Orleans, La., reported a reduction from 60 to 30 per cent primarily because of the institution of Wangensteen drainage, but he said the mortality in his department was now increasing because of the false security.

Dr. Claude J. Hunt, Kansas City, Mo., **Pancreatic Cyst:** Historical data and the classification of pancreatic cysts were presented. It was emphasized that the cyst may present at varied positions with relation to the stomach. Symptoms are due to pressure and disturbed physiology. The x-ray should be of significant aid in establishing the tumor as arising from the pancreas. The case presented was treated by excision of the cyst, although this is by no means always possible.

Dr. Frank C. Mann, Rochester, Minn., **The Effect of Decreasing the Lumen of a Blood Vessel:** Various methods of measuring the rate of flow of blood through a blood vessel were discussed, paying particular attention to the method utilizing dissemination of heat. Unexpected results were obtained so far as considerable

decrease in the lumen of the artery could be effected without producing a decrease in the blood flow. The external diameters can be decreased as much as 50 per cent with a resultant decrease in flow of only 18 per cent.

Dr. Everett P. Coleman, Canton, Ill., **Injection of the Right Stellate Ganglion with Alcohol in Paroxysmal Tachycardia:** The stellate ganglion of a patient who had daily attacks of paroxysmal tachycardia associated with a diseased gallbladder was injected with alcohol. The history of cholecystitis was of twenty years' duration and the paroxysmal tachycardia of four years' duration, suggesting that there was little relationship between the two diseases. The injection of the alcohol produced almost an instantaneous cessation of the paroxysmal tachycardia, the heart rate dropping from 200 down to 88. The dangers associated with the injection were discussed.

Dr. Gordon S. Fahrni, Winnipeg, Canada, **Hyperparathyroidism:** Four patients with hyperparathyroidism were reported, paying particular attention to the author's last case in whom two tumors were encountered. In this patient one tumor was found embedded in a lobe of the thyroid; whereas, the other was found in the mediastinum. X-ray pictures of the bones were shown to illustrate the calcification occurring in the decalcified areas following excision of the tumor. The importance of the laboratory data was stressed.

Dr. William T. Peyton, Minneapolis, Minn., **Hemangioma and Its Treatment:** The author emphasized that many cavernous hemangiomas need not be excised but could be eliminated by the injection treatment. The injection treatment recommended consisted of several injections of not more than 3 c.c. of sodium morrhuate into the tumor. A comparison of results in patients treated with radiation and those treated with injection revealed a cure in about 55 per cent and 82 per cent, respectively.

REVIEW OF THE SOUTHERN SURGICAL ASSOCIATION MEETING, DEC. 7-9, 1937, BIRMINGHAM, ALA.

HOWARD R. MAHORNER, M.D., NEW ORLEANS, LA.

(From the Department of Surgery, Tulane University School of Medicine)

THE fiftieth annual meeting of the Southern Surgical Association was held in its nativity city, Birmingham, Ala., at the Tutwiler Hotel, December 7, 8, and 9. One hundred forty-nine members were present. Of forty papers on the program thirty-eight were presented.

Dr. Isidore Cohn, New Orleans, La., discussed malignant disease of the breast in negroes. At the Charity Hospital in New Orleans from 1932 to 1936 inclusive there were 408 cases of carcinoma of the breast in white and colored races. Two hundred and fifty-six of these were in the colored race. The incidence of 1.7 per 1,000 admissions has been a fairly constant rate during this time without annual variation. The average age for the colored patient was 4.7 years younger than was average age of the whites. The average duration of symptoms was 1.6 years. Of 224 colored patients in whom the records were sufficiently complete for such study, 118 had radical operations. Sixty-three of these had glandular involvement; that is, 60 per cent had axillary involvement on admission. The mortality after operation is approximately 65 per cent in from one to four years.

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Dr. Hugh Trout, Roanoke, Va., showed a beautiful colored moving picture of the radical operation for carcinoma of the breast. The operation he uses is essentially the Halsted operation. He believes that irradiation should be used as an adjunct to surgery but that it has no replacement value for surgery.

Dr. Edgar L. Gilcreest, San Francisco, Calif., read a paper on lumbosacral and sacroiliac sprains, calling attention to the fact that back pain as a single entity probably causes more industrial disability than any other condition. The causes of back pain are numerous. Acute or chronic sprain is the most frequent cause. Infection is the next most frequent cause. Congenital anomalies, neuropathic condition, metabolic conditions, and neoplastic causes are less frequent. Protrusion of the intervertebral disk is a possibility which must be ruled out. Dr. Gilcreest paid particular attention to acute and chronic sprains and slips in the sacroiliac and lumbosacral regions. He outlined the clinical aspects and advocated heat and massage and manipulations, which consist of forcibly flexing the knee with counterforce on the shoulder while the patient is on his side, and flexing the leg with the patient on his back, the other leg hanging over the side of the table fixing the pelvis. Relief is prompt in a large percentage of patients after these measures.

Dr. Ralph G. Carothers, Cincinnati, Ohio, discussed internal fixation of fractures of the neck of the femur. Dr. Carothers had some helpful suggestions for doing the operation. He uses the Austin Moore method with variations. A towel clip at the middle of Poupart's ligament serves as a very helpful guide to the head of the femur. After reduction, anteroposterior roentgenograms with this clip in place and a lateral roentgenogram with the edge of the casset flat on the table give information in the former view of the accuracy of the towel clip as a guide and in the latter view of the inclination from the horizontal necessary to hit the head of the femur. Of 26 cases in which he used this method, 9 had intertrochanteric fractures and 17 had intracapsular fractures. Of the intertrochanteric fractures 8 had bony union and 1 died. Of 17 intracapsular fractures, 2 died early; 5 have bony union in good position; in 4 the neck was absorbed; 2 had union but also a limp; 1 died of pneumonia ten weeks after operation; 3 have been operated upon less than three months.

Dr. R. Arnold Griswold, Louisville, Ky., discussed the treatment of delayed union and nonunion of fractures by subcutaneous drilling. In 12 cases in the last two years, all of which had delayed union or nonunion of subcutaneous bones, Dr. Griswold performed multiple drillings in a fan-shaped manner through the two fragments. After this procedure, all obtained union. Dr. Griswold called attention to the fact that this relatively minor operation will often obtain the desired results without having to perform more radical surgery.

Dr. Frank Strickler, Louisville, Ky., attempted to find a more suitable material for internal fixation of the fractures. Chromicized beef tendon, which he has prepared specially, was found to be a very suitable material. It has a certain rigidity, but it is absorbed after approximately ninety days. It doesn't show in the x-rays. He showed various methods of using this material for fixation of fractures; as a cuff over the two fragments held by kangaroo tendon, as two pegs for intermedullary support, and other methods.

Dr. J. C. Gerster, New York, N. Y., reported a case of rupture of a diverticulum of the jejunum resulting in peritonitis. The patient's general condition permitted only drainage of the diverticulum. Death resulted later in the con-

valescence from uremia. Other than Meckel's diverticulum, diverticulosis of the colon is most common, next diverticulosis of the duodenum, and lastly single and multiple diverticulosis of the jejunum. The author's case had nine diverticuli, one of which had ruptured. From the literature he collected 186 cases in 129 references. A large percentage of diverticula of the jejunum give no symptoms. Others give flatulent dyspepsia and others symptoms of chronic intestinal obstruction. Occasionally rupture occurs.

Dr. Vernon C. David, Chicago, Ill., read a most instructive paper on the influence of diverticulitis on the mortality associated with surgery of the colon. Numerous complications occur from diverticulitis of the colon. Adhesions resulting from diverticulitis may seriously interfere with resection of the colon for carcinoma. Inflammation of sigmoid diverticula may result in adhesions to adjacent organs, notably the bladder. Appendices epiploicae may contain diverticula and for that reason are not to be ligated. In 5 instances he has noted abdominal wall infections from perforation of a diverticulum on the loop of a colostomy. In his experience perforation of a diverticulum resulted once from colonic irrigation, twice from obstruction. In 2 instances he found pylephlebitis caused by diverticulitis.

Dr. Henry W. Cave, New York, N. Y., discussed the surgical treatment of intractable chronic ulcerative colitis. In those instances in which medical measures have failed and the disease has persisted in a severe and intractable form, the only hope of cure lies in eradication by total, or in the segmental form by partial, colectomy. In his experience appendecostomy cecostomy, and colostomy have no place in the treatment of this disease as they are intended to permit irrigations which are not effective. After careful preparation of the patient, colectomy is performed in three or four stages. Ileostomy as the preliminary step is essential. Five weeks later, colectomy, removing the ascending, transverse, descending, and part of the sigmoid colon. Two to six months later combined resection of the remaining colon and anus is performed. He has performed 6 colectomies with no deaths. After colectomy the terminal ileum assumes the functions of the colon to a certain extent and the discharges become less fluid, but this transition of function in the ileum occurs only after the colon has been removed.

Dr. John Gerster, New York, N. Y., in discussing Dr. Cave's paper, said that one of his patients had had a cecostomy for ten years and wanted this cecostomy closed as the ulcerative colitis seemed to be dormant. X-ray, however, showed a colon with the lumen almost obliterated, the barium showing in parts as only a thin line.

Dr. Fred Rankin, Lexington, Ky., president of the Association, discussed this paper in some detail. He called attention to the fact that ileocolostomies and subtotal colectomies are rarely indicated because segmental involvement is rare.

Dr. Arthur W. Allen, Boston, Mass., gave a splendid paper on carcinoma of the stomach with special reference to total gastrectomy. At the Massachusetts General Hospital there have been 713 cases of carcinoma of the stomach. One hundred and seventy-six of these were subjected to subtotal gastrectomy and 13 to total gastrectomy. Two additional cases had total gastrectomies for other reason than carcinoma. He advocates anastomosis of the long loop of jejunum to the end of the esophagus, anchoring the jejunum to the diaphragm, an enterostomy at the dependent portion of the loop of jejunum, and a jejunostomy distal which is brought out of a side stab wound for feeding. In this series of 15 patients, 7 are dead; 2 are alive without recurrence, 1 twelve months and 1 four years after operations.

Dr. Edward H. Ochsner, Chicago, Ill., reported an interesting case of simple nonparasitic cyst of the liver. Such cysts are extremely rare, are more common in women, and occur at the anterior margin of the lower surface of the liver. In his case the cyst of the liver contained four gallons of murky fluid. The wall was partially removed at operation and the remaining wall was marsupialized. Microscopic section showed columnar epithelium. The patient made an uneventful recovery and at a subsequent operation for another cause some years later there was no evidence of the cyst except adhesions from the region of the liver to the abdominal wall.

Dr. R. L. Rhodes, Augusta, Ga., discussed acute cholecystitis. He believes that more attention should be paid to an analysis of the blood chemistry in these cases. Blood sugars, blood chlorides, and nonprotein nitrogen and uric acid determination are all important and preliminary preparation of the patient should be directed in some measure to elevation of lowered blood sugar and chlorides and to reduction of elevated nonprotein nitrogen. In discussing this paper, **Dr. Charles Gordon Heyd,** New York, N. Y., emphasized the necessity of reestablishing diminished liver function. He believes that drainage of gallbladder facilitates drainage of the liver.

Dr. William B. Marbury, Washington, D. C., discussed retroperitoneal (retrocolic) appendix. The condition is not extremely common, but it does occur where the appendix in its embryonic descent gets caught under the colon and becomes entirely retroperitoneal. The clinical appearance is apt to differ from that of typical acute appendicitis in that the pain is not localized definitely at McBurney's point. The tenderness is higher and more posterior. Blood or pus may be found in the urine. Dr. Marbury reported 6 cases. The operation is sometimes difficult because the appendix is hard to find. It may be traced by first identifying its base at the ends of the longitudinal bands on the cecum.

Dr. Vernon Mastin, St. Louis, Mo., in an extensive study based on records of 9,592 patients having operations showed that preoperative medication definitely influenced the incidence of postoperative complications. Of 7,901 abdominal operations, 5,324 received morphine and atropine preoperatively; 2,318, morphine and hyoscine; and 259, morphine and barbiturate. In the morphine and atropine group 2.7 per cent developed cystitis or pyelitis; in the morphine and hyoscine group, 13.7 per cent; and in the morphine barbiturate group, 2.3 per cent. Studies for other operations and complications showed similar incidences. Morphine and a barbiturate administered preoperatively is followed by fewer complications than the other two combinations.

Dr. R. Glen Spurling, Louisville, Ky., has sectioned the scalenus anticus muscle in 20 cases for neurocirculatory compression without cervical rib. Of 17 cases which he was able to follow, relief of symptoms had been complete in all except 3. In these 3 there had been improvement. The symptoms of this syndrome, which was first recognized by Naffziger as occurring independently of supernumerary rib, are pain in the distribution supplied by the brachial plexus, numbness or hyperesthesia, muscular weakness, diminution in pulse, especially when the patient turns the head to the affected side and takes a deep inspiration, and tenderness over the scalenus muscle. The condition is not rare and better appreciation of the symptoms and diagnostic signs will result in more cases being recognized.

Dr. Charles Bagley, Jr., Baltimore, Md., reported 2 cases of gas bacillus infection causing brain abscess. He found only 20 proved cases of this type in the literature. One of his cases was due to a puncture wound, the second to

a compound fracture resulting from a blow with a stick. Both recovered. One, however, developed tetanus during convalescence, and under huge doses of serum and avertin to control convulsions the patient recovered.

Dr. Mims Gage, New Orleans, La., gave a very complete discussion of pilonidal sinus. Differentiating it from sacrococcygeal dimple, he discussed the various etiologic possibilities and why the evidence is greatest in favor of the condition being a remnant of the neurenteric canal. Among important points in the treatment of the condition are accurate localization of the sinus by lipiodol injection and x-ray and by preliminary injection of the sinus on the fifth, fourth, and third days preceding the operation with methylene blue to stain the tract. The sinus is excised completely and primary closure is made, obliterating the dead space with sponge pressure. All 42 cases healed without recurrence.

Dr. L. Wallace Frank, Louisville, Ky., discussed thoracoplasty in the treatment of tuberculosis with special reference to the Semb procedure. Dr. Frank believes that the Semb operation is an epochal advance in the treatment of tuberculosis as it permits release of the apex and closure of cavities with resection of a minimum number of ribs, thus preserving more of the normal lung and accomplishing with greater certainty closure of cavities than by other methods of thoracoplasty.

Dr. Byrd C. Willis, Rocky Mountain, N. C., reported 104 cases of empyema in children occurring in years 1915 to 1937. There were 5 deaths. There were 79 cases under eleven years of age with 4 deaths. All cases are treated by interrib puncture with trocar and insertion of a tube. Careful attention to the after-care is necessary, being sure that drainage continues satisfactorily by persistent irrigations. No chronic empyemas developed. The procedure has the advantage of being a relatively simple method of obtaining satisfactory drainage. A minimum scar is left.

In discussing this paper, **Dr. James Mason,** of Birmingham, Ala., said he had employed rib resection on a similar group of cases with 1.6 per cent mortality. Other discussions showed differences of opinion on the method of obtaining drainage, but all agreed that different methods with careful attention to detail would accomplish satisfactory end-results. In discussing this paper, **Dr. George A. Hendon,** of Louisville, Ky., stated that he had used effectively warm air passed over iodine with which to irrigate the empyema cavity.

Dr. James D. Rives, New Orleans, La., read a paper on pulmonary abscess and discussed the factors influencing mortality. In 239 cases occurring in Charity Hospital from 1929 to 1936 and at Touro Infirmary from 1926 to 1936, there were 100 deaths. The mortality was higher before ten (52 per cent) and after forty years of age. The known mortality in this series is 42 per cent. One hundred and ninety-six of the cases were treated medically with a mortality of 44 per cent and 43 per cent were treated surgically with a mortality of 33 per cent. He does not believe duration of abscess is an important factor in the mortality. However, the amount of lung involvement is a very important factor. Single abscesses resulted in the 28.5 per cent mortality; bilateral abscesses, in 93 per cent; multiple abscesses in a single lobe, in 57 per cent; and abscesses in multiple lobes in one lung, in 45 per cent mortality. Of the 100 fatal cases only 23 cases had extrapulmonary and extrapleural complications. He believes that surgical treatment is indicated in abscesses located peripherally and in cases which do not respond to conservative measures. Aspiration of the abscess and artificial pneumothorax have no place in the treatment of this condition.

Dr. Addison G. Brenizer, Charlotte, N. C., demonstrated excellent results obtained by the use of an original method of fascial implants in the repair of harelip and cleft palate.

Discussing Dr. Brenizer's paper in which Langenbeck's operation was mentioned, **Dr. H. J. Boldt,** New York, N. Y., treated the assembly to some historical reminiscences. When he was very young, having suffered a broken arm, he was a patient of the great Langenbeck. He stated that in convalescence Langenbeck each day had him go to the abbatoir's and take two types of baths, the so-called "blood bath" in which he immersed his arm in a bucket of fresh beef blood for half an hour, followed by an "intestinal bath" in which he bathed his arm in the opened abdominal cavity of a freshly killed beef for half an hour. He thought it would be interesting for the members to know how the old methods contrast with the new.

Dr. J. Barrett Brown, St. Louis, Mo., showed beautiful results obtained by grafts in surface defects of the hand. Full thickness grafts were more frequently used; occasionally a pedicle graft was used. Careful dissection of scarred tissue and meticulous attention to detail were stressed.

Dr. Lawrence R. Wharton, Baltimore, Md., described a new technique of reconstructing the vagina and reported its successful use in 4 cases. The technique consists of making a stent, or mold, of balsam wood covered by rubber which is inserted after dissection between the rectum and bladder in the normal location of the vagina. The stent is left in for approximately three weeks and epithelium grows in from the surface to completely line this new cavity which retains its adequate size. Microscopic sections of the wall of these patients have been taken and are found to be entirely healed over with stratified squamous epithelium.

Dr. R. L. Sanders, Memphis, Tenn., discussed cysts of Gartner's duct. He reported a case of a large cystic mass occurring in the anterior vaginal wall and protruding from the introitus which initially looked like a prolapse but proved to be a cyst in the anterior vaginal wall. It extended on over to the wall of the bladder, was successfully removed at operation, and contained a thin mucilaginous fluid. Its location and pathologic characteristics proved it to be a cyst of Gartner's duct.

Dr. William T. Black, Memphis, Tenn., reported 3 cases of hernia through the posterior vaginal wall. This is a true hernia, has a sac, and is to be differentiated from rectocele, with which it might be confused to the great regret of a surgeon. One of his patients was nulliparous; 1 case occurred after trauma. Treatment consists in isolation of the sac, excising it and repairing the defect.

Dr. W. D. Haggard, Nashville, Tenn., in discussing this paper, stated that he had had one such case which he had cured by opening the sac and packing the culdesac from below with gauze, thus producing adhesions which cured the hernia.

Dr. William E. Lower, Cleveland, Ohio, reported an interesting case of diverticulum of the urethra in a woman. Such a possibility is to be kept in mind in instances where there is prolonged urinary frequency or dysuria. Diagnosis is made by urethroscopy, probing the diverticulum, and injection of a radiopaque medium. Treatment consists in complete excision and primary closure of the urethra.

In discussing this **Dr. Guy Hunner,** of Baltimore, Md., reported 3 cases, showing slides of each, in which a stone was found in a diverticulum of the urethra.

Dr. Curtis Tyrone, New Orleans, La., read a paper on the comparative study of three types of hysterectomy. Of 764 hysterectomies performed by the late great **Dr. C. J. Miller** and himself in the years 1930 to 1936, inclusive, 316 (40 per cent) were supravaginal hysterectomies; 137 (20 per cent), complete abdominal hysterectomies; 311 (40 per cent), vaginal hysterectomies. The causes for hysterectomies were fibroids in 382 cases; fibrosis and chronic metritis in 238 cases; and pelvic endometritis in 74 cases. Other causes were present in the remaining cases. The operative time is no factor in determining which operation to use, because the difference in the average time necessary to perform each of the three types of operations is not greater than five minutes. He reported the commendable mortality of 1.9 per cent for supravaginal hysterectomies; 2.2 per cent for complete abdominal hysterectomies; and 0.64 per cent for vaginal hysterectomies. Combined mortality was 1.4 per cent.

Dr. Walter E. Dandy, Baltimore, Md., reported a case of an intracranial aneurysm which he first saw soon after its initial symptoms. Exploration revealed a small aneurysm of the circle of Willis, which had ruptured and formed a small false sac and which was pressing on the third nerve. He put a silver clip around the neck of the aneurysm sac and fulgurated the aneurysm itself. Complete cure resulted. Not all aneurysms at this location are amenable to surgical treatment because of (1) the difficulty of diagnosis and (2) the difficulty of approach of certain arteries.

Dr. Rudolph Matas, New Orleans, La., discussed Dr. Dandy's paper. He reviewed in great detail the problem of aneurysms of the circle of Willis and stated that this was the first true arterial aneurysm of the circle of Willis which had been cured by occlusion. He believes that cranial encephalography opens great possibilities for diagnosis of these conditions.

Dr. Waltman Walters, Rochester, Minn., reported 7 cases of suprarenal cortical tumors in which operation had been done and in which recovery followed. He emphasized the difficulty of differentiating between adrenal cortical tumors and pituitary basophilism. Adrenal cortical tumors may mask under clinical characteristics similar to those of pituitary basophilism and, therefore, doubtful cases should have the advantage of an exploration of the adrenal glands. Not all tumors of the adrenal glands produce typical virilism or changes in the secondary sex characteristics. In some instances the changes are very mild and because these tumors tend to be malignant early diagnosis is imperative. Recovery in 7 consecutive cases he attributes to increased knowledge of the disturbed physiologic processes and to better postoperative care. This consists of administration of large amounts of sodium chloride and sodium citrate and the daily administration of an adequate amount of cortical hormone in the immediate postoperative period.

Dr. Loyal Davis, Chicago, Ill., discussed the surgical problem of hypertension. Dr. Davis' indications for surgery in essential hypertension are extremely rigid. He accepts only those cases which have a potentially reversible blood pressure and which, on the other hand, do not respond to medical management and administration of cyanate. The pressure must be above 200 systolic and 100 diastolic and there must be no evidence of renal impairment due to glomerular damage. Thus, he has had a small number of patients in which he has done a combined sympathetic and splanchnic resection by the transthoracic method advocated by Peet. Of 6 patients operated upon, all have returned to their preoperative blood pressure levels within ten days. On the other hand, he had another patient who was operated upon elsewhere and after returning to him, though the pressure had reached its

previous level, the patient responded to cyanate therapy. In an additional case of his own the patient became sensitive to cyanate therapy after operation. Although the operation may not accomplish all that has been hoped, it may change the patient's response to cyanate, thus opening new possibilities for alleviating the condition.

Dr. George Crile, Cleveland, Ohio, prefers to remove the hypogastric plexus for essential hypertension. He has found operations on the sympathetic system helpful in neurocirculatory asthenia, recurrent hyperthyroidism, and hypertension and such vascular conditions as Raynaud's disease. He considers the adrenals and hypogastric plexus together as an organ which is an energy arsenal for use in emergency. As might be suspected, the lion, which has the most tremendous burst of energy, has relatively the largest adrenals and hypogastric plexus. In 213 cases of hypertension, after removing the hypogastric ganglia, at least symptomatic benefit has resulted in approximately 90 per cent of the cases.

Dr. Howard R. Mahorner, New Orleans, La., discussed the modern treatment of varicose veins. In a test devised by himself and **Dr. Alton Ochsner,** New Orleans, La., it is possible to determine the exact level of leaks from the deep to the superficial system in the varicosed extremity. In those instances in which the valves of the saphenous vein alone are incompetent, high ligation of the saphenous with injection of a sclerosing solution in the distal stem results in a cure. However, in those instances in which the comparative tourniquet test shows incompetence of valves of the communicating veins as well as incompetence of the valves of the saphenous, not only high ligation of the saphenous is done, but also low ligation to reduce the retrograde flow from the deep to the superficial system. A moving picture demonstrating the test and treatment was shown.

Dr. Hugh Gamble, Greenville, Miss., reported a new operation for tracheoesophageal fistula and reported a case of tracheoesophageal fistula occurring in the newborn. He divided the stomach at the cardiac end, closed the distal part, and made a gastrostomy. The proximal end, which was attached to the esophagus, was brought out through the abdominal wound permitting free drainage of the esophagus. The patient lived for fifteen days and died of peritonitis due to a rupture of an ulcer of the stomach.

Dr. K. H. Aynesworth, Waco, Tex., discussed pain in acute abdominal surgical emergencies. After reiterating the various theories relative to the transmission of pain from abdominal organs, evidence was advanced for accepting the theory that pain is transmitted by cerebrospinal nerves and not by sympathetics. Anatomic structure and disturbed physiology determine the type of pain, and the character and location of the pain are helpful guides to the correct diagnosis. The relationship of local and referred pain to various pathologic processes in the abdomen was considered in detail.

Dr. W. O. Bullock, Lexington, Ky., advocated the use of glass rods over which mattress sutures are tied to prevent disruption of a laparotomy wound in cases which have to be drained.

Dr. Charles R. Robins, Richmond, Va., presented an operation for the cure of direct inguinal hernia. Dr. Robins drew attention to the fact that that part of a conjoined tendon which is usually attached to Cooper's ligament is frequently deficient in patients with indirect inguinal hernia. Dr. Robins' operation consists of inserting a fascial suture, beginning at the firm suprapubic ligament, catching the edge of the rectus fascia, sewing it down over the cord to Poupart's ligament,

continuing in this plane suturing the fibers of the internal oblique and transversalis muscles to Poupart's ligament. The aponeurosis of the external oblique is sutured over this. This supplants the deficiency in the conjoined tendon in these cases.

Dr. Carrington Williams, Richmond, Va., advocated the use of an abdominal approach in the repair of inguinal hernia; (1) in cases with strangulated hernias, (2) where it is desirable to remove the appendix at the same operation, (3) for sliding hernias, (4) for congenital hernias, and (5) for cases in which there is an undescended testicle. The usual oblique incision is made, but after incising the aponeurosis of the external oblique a split-muscle incision is made above the level of the internal ring. The abdominal cavity is opened by incising the peritoneum and the neck of the hernia lies immediately below this approach. The peritoneum is cut across posterior to the hernial orifice and the abdominal cavity is closed by suturing the anterior and posterior layers of the peritoneum. The hernial sac is then dissected out. This permits a good exposure, especially for such conditions as present a potential resection of the bowel or omentum. In the congenital hernia it is not necessary to remove the sac. Closure may be made in any one of the acceptable ways of repairing hernial defects in this region.

Dr. E. H. Adkins, Miami Beach, Fla., reported a case of a male, twenty-five years of age, who after lifting had a sharp pain in the abdomen. Careful study revealed preoperatively a diverticulum of the descending colon. Exploration, however, revealed a spleen which was entirely below the level of the umbilicus, the lower edge of which was in the pelvis. Splenectomy resulted in cure.

Among the high lights of the meeting were: an address by **Dr. Irvin Abell,** Louisville, Ky., characterized by symphonic eloquence and having all the profundity and high-mindedness of a prayer by Samuel Johnson, and the presidential address by **Dr. Fred Rankin,** Lexington, Ky., paying tribute to the early history of the organization and touching in an illuminating manner many of the successes on the road of surgery.

Book Reviews

Diseases of the Nervous System in Infancy, Childhood and Adolescence. By Frank M. Ford. Pp. 953, with 107 illustrations. Springfield, Ill., and Baltimore, Md., 1937, Charles C. Thomas, Publisher. $8.50.

This is an accurate and comprehensive text, perhaps somewhat voluminous (953 pages) but almost necessarily so if the subject is to be presented well and brought up to date, as the author has done. Illustrative case histories throughout the text comprise approximately 100 pages. The reviewer personally feels that case histories are unnecessary in a well-written text, such as this one is, and therefore that the book might have been shortened, to the extent of omitting these pages, without detracting from its subject matter.

The first two chapters (148 pages) deal with the examination of the nervous system and the clinical aspects of the anatomy and physiology of the nervous system. In the succeeding chapters the author presents the diseases of the nervous system according, as he states in the preface, to a classification based primarily upon etiology with due emphasis upon clinical features, rather than upon a purely anatomic classification as is customary. This classification improves one's understanding of these diseases. The individual diseases are discussed both intelligently and intelligibly.

Following the discussion of each disease entity is a brief but carefully chosen bibliography. In the selection of the references the author favored those which are written in English and which are easily available.

There are not many cuts for so complete a book but those pictured are clear and illustrative. The print, paper, and binding are all of very good quality.

The book is a medical rather than a surgical treatise; hence it is of most value to the neurologist and pediatrician, but valuable also to the neurosurgeon and in fact to any doctor of medicine. The reviewer, a neurologist, thoroughly enjoyed the book and commends the author upon his excellent work.

Genital Abnormalities, Hermaphroditism, and Related Genital Abnormalities. By Hugh H. Young. Cloth. Pp. 649, with 379 illustrations. Baltimore, 1937, The Williams and Wilkins Company. $10.

The scope of this interesting and valuable volume is not fully indicated by its title, since it covers not only hermaphroditism and pseudohermaphroditism and abnormalities of the male genitalia, but also disturbances of the endocrine glands which influence the sex organs and characteristics. It contains both a thorough-going review of the literature and a detailed discussion of Dr. Young's extensive experience. It is profusely and excellently illustrated.

The discussion of hermaphroditism in literature and art, the review of the embryology of the condition, and the engaging presentation of case histories of hermaphroditism and the much commoner pseudohermaphroditism make it an abbreviated counterpart of Neugebauer; but the discussions of the rôle of the endocrine glands in the production of these and related conditions, as well as the admirably illustrated descriptions of the operations required for their correction, make the scope of this book much greater than that of Neugebauer's.

While hermaphroditism and pseudohermaphroditism are rare conditions, they are of such importance to their victims that every physician should familiarize himself with them both so that he can recognize them and in order to familiarize himself with the possibilities of plastic surgery in enabling the patient to lead the satisfactory sexual life which the disorder usually denies him.

In addition Young discusses his experience with the "adrenogenital syndrome" in which abnormalities of the genitalia and sex characters have resulted from hyperplasia or neoplasms of the adrenal cortex and describes his operation for simultaneous exposure of both adrenals, which he advocates to obviate the disastrous results of removal of a solitary gland.

Allied conditions such as arrhenoblastoma of the ovary, hyper- and hypogenitalism, and gynecomastia are reviewed, and the relations of the various endocrine glands to the sexual organs and characteristics are reviewed. These are topics of lively interest today, and it is to be hoped that such investigations as this, coupled with those of the experimental endocrinologist, will lead at some future time to the possibility of understanding them so thoroughly that exploratory operations will become unnecessary.

The author also reviews the literature, discusses his own experience, and describes in detail the operative treatment of hypospadias, epispadias, exstrophy of the bladder, cryptorchidism, congenital valves of the prostatic urethra, and atresia ani urethralis.

This book ought to be in the hands of every urologist; the general and plastic surgeon and the internist will find much of value and even of entertainment (the case reports of pseudohermaphroditism) in it.

The Therapeutic Problem in Bowel Obstructions. By Owen H. Wangensteen. Pp. 360, with 90 illustrations. Springfield, Ill., 1937, Charles C. Thomas. $6.

Few conditions in medicine have more provokingly incited the clinical and experimental investigator than the subject of acute intestinal obstruction. That this problem thoroughly deserves such perennial inquiry and resolute research is made obvious by the lamentable knowledge that until relatively recently the mortality attending this serious condition has not been materially reduced in over a half century. However, it can be stated with gratifying encouragement that during the past decade the lights from the experimental laboratory have begun to dispel the Stygian clouds of obfuscation overhanging this grave and perplexing problem. Because no light has been more brilliant or more piercing than that of Wangensteen and his coworkers, a monograph on this subject by this investigator is eagerly received.

The author's deserving reception of the Samuel D. Gross Prize award has effected the publication of this compendium. Briefly, it is a concise elaboration of the more important aspects of the therapeutic considerations in bowel obstruction in the light of the writer's investigations and experience in this particular endeavor.

The first portion of the text is a perspicacious exposition of the physiologic effects and pathologic consequences of bowel obstruction and distention, the criteria for its early recognition, and a critical analysis of the therapeutic value of various procedures and agents. On the basis of extensive clinical and experimental investigations, the author clearly demonstrates the rational efficacy of saline solution in high obstruction (rather than low) where fluid loss is predominant, the judicious utilization of blood transfusion in strangulated obstructions, the expedient value of conservative decompression by suction applied to a duodenal tube, and the discerning indications for operation.

The remainder of the text is concerned primarily with a rather classic description of the various types of intestinal obstructions caused by congenital atresia, imperforate anus, obturation, Hirschsprung's disease, hernia, volvulus, intussusception, etc. Whereas this is little more than a review of the accepted facts characterizing each entity, its lucid presentation makes it a valuable addition. The text is adequately illustrated with appropriate and well-reproduced drawings and photographs. The bibliography following each section, although not very extensive, contains the more significant and pertinent references.

Whereas this monograph purposes "to set forth in a concise manner the important aspects of the therapeutic problem in obstruction of the bowel as they appear to the writer," actually it represents the recent progress and the most important contributions yet made in the management of this grave malady.

Collected Papers of the Mayo Clinic and Mayo Foundation, Volume XXVIII, 1936. Edited by Richard M. Hewitt, Lloyd G. Potter, and A. B. Nevling. Ed. 1. Cloth. Pp. 1331, with 212 illustrations. Philadelphia, 1937, W. B. Saunders Company. $12.

Collected from many sources of publication, these papers do a real service to the reading medical public in that they not only summarize the year's productive work in the Rochester clinic but in that, in many instances, they give authoritative and up-to-the-minute information on the medical questions of the day. Covering a wide range of subjects, they offer great interest to the general practitioner and to the specialist. "What happens to gastric acidity following operations for gastric and duodenal ulcer?" "Why do some gastroenterostomies fail to function?" These and many other questions are answered in 90 articles appearing in full, in 50 by abridgment, in 80 by abstract, and in 50 by title. A few less than 300 contributors offer this material. Of unusual interest is Mann's work on "Hepatic Physiology and Pathology" and the experimental work done in his laboratory on this and other subjects.

Almost all of the subject material in this volume concerns itself with applied science as related to the practice of medicine. High lights are such subjects as, "Studies on the Influence of Certain Drugs in Relation to Biliary Pain," "The Treatment of Infections of the Urinary Tract," "Reactions Following Operation for Hyperthyroid Conditions," "Postoperative Effects of Pneumonectomy," and many others of similar nature. Dr. W. J. Mayo contributes a mellow reference to "Libraries Useful in Their Day." In short, this volume can be heartily commended to anyone interested in clinical medicine and a study of its problems. There are but few who would fail to gain considerable knowledge from its perusal.

Textbook of Diagnostic Roentgenology. By Lewis J. Friedman. Pp. 623, with 638 illustrations. New York, 1937, D. Appleton-Century Co. $10.

The need for general treatises on roentgen diagnosis is still apparent. This volume presumably fills a place between the brief texts for students and the more elaborate, detailed reference books.

With a wealth of clinical material at his disposal, the author has produced a very comprehensive survey of the subject. All phases of roentgen diagnosis are

covered, including the physical principles, methods of technique, and dark-room procedures. There is, therefore, a considerable material between the covers of this one book.

The scope of roentgen diagnosis is so extensive that a complete presentation cannot be adequately made in so limited a space. As a result, many subjects are treated rather superficially. There is, perhaps, too much emphasis on the rarer diseases and the more bizarre roentgen manifestations. For example, bone tumors and infections are treated rather briefly while considerable space is devoted to the rare lesions, such as leprosy, osteopoikilosis, etc.

Certain features are open to specific criticism. In the fairly long chapter on fractures, nothing is stated as to methods of determining whether a proper reduction has been made. The complete separation of acute from chronic osteomyelitis seems hardly justifiable. The use of the term "tubercular" for "tuberculous" will no doubt be corrected in later editions.

The material on intestinal obstruction is open to considerable question. The author apparently prefers to give barium by mouth rather than by enema in suspected cases, advice which will not be concurred in by most surgeons and roentgenologists. The impression is created that ileocecal intussusception is rare and that only occasionally it can be diagnosed roentgenologically. Neither of these statements is sound.

While other errors of a similar nature could be pointed out, the material on the whole is sound and most of the points in dispute are not serious.

The illustrations are well selected and rather well reproduced. A bibliography is included but it is undoubtedly not intended to be complete. The book is well indexed and well printed.

The treatment accorded most of the subjects is too brief to satisfy the specialist in roentgen diagnosis. While it may have more value for other specialists, the same objection still obtains. To the general practitioner, however, this book should have considerable appeal. It enables him to obtain a brief and reasonably authoritative statement concerning almost any problem in which roentgen diagnosis is a factor. The concise and clear exposition of physics and technique will also be of value to this group. It is recommended as a valuable addition to the library of the physician practicing general medicine.

The Collapse Therapy of Pulmonary Tuberculosis. By John Alexander. With 679 illustrations. Springfield, Ill., 1937, Charles C. Thomas.

When Dr. Alexander's first book, *The Surgery of Pulmonary Tuberculosis*, appeared in 1925, the surgical treatment of pulmonary tuberculosis was in its infancy throughout the world and particularly so in this country as compared to those of Central Europe. Due to a large extent to the excellence of this first book, the procedures which were discussed were rapidly adopted in America and the increase in their use has been phenomenal. A number of large sanatoria have found that some form of collapse therapy is indicated in approximately 75 per cent of their patients. The advances made during the past twelve years have more or less compelled Dr. Alexander to rewrite the entire book, except for those parts dealing with historical matters. In addition the illustrations have been almost entirely replaced and added to so that there are 367 figures that contain 679 separate illustrations. The size of the book is three or four times that of the first and the bibliography has increased from approximately 500 references to more than 1,300.

The reader will find that the book has been written for the internist, the surgeon, and the roentgenologist. It is based upon an experience with the surgical treatment of approximately 1,800 patients with pulmonary tuberculosis. The surgeon will be particularly interested in the chapters dealing with the various types of operations that may be used in obtaining collapse of the diseased lung. The indications for and the contraindications to ten such operative procedures are considered in detail. Each procedure is classified by adequate illustrations, consisting of line drawings together with serial x-rays which show the preoperative and postoperative condition of the lungs. The description of the operative technique is in most instances given in great detail. Possibly the Semb procedure and the employment of electrosurgery for the division of adhesions deserve fuller consideration. The desirable and undesirable features of each operative procedure are impartially weighed and the interrelationship of the various procedures is discussed. A consideration of the different types of tuberculous empyema is particularly instructive as is also the chapter dealing with tuberculosis of the thoracic wall.

Practically all of the book, except for the parts dealing only with operative technique, will be of interest to the internist. The choice of patients for collapse therapy and the choice of procedure for the particular patient are given careful attention. Various types of pulmonary and pleural lesions are discussed from the viewpoint of the choice of the proper treatment. The causes of failure as well as success in the care of patients are fully considered. Of particular interest to the internist are the excellent chapters by Dr. John Blair Barnwell on pneumothorax and by Dr. Kirby Smith Howlett, Jr., on oleothorax. These subjects are thoroughly and carefully considered.

The book will be of interest and help to the roentgenologist who works with and advises the surgeon and internist. The confusing shadows which may be observed after operative procedures are discussed. The findings on fluoroscopic examination before and after various forms of treatment are described.

Of interest to all will be the two excellent chapters by Dr. Max Pinner on "Physiological Principles of Collapse Therapy" and the "Pathology of Pulmonary Collapse."

The book can be recommended without reservations as an authoritative treatise of the subject.

Neurology. By Roy R. Grinker, Chairman, Department of Neuropsychiatry, Michael Reese Hospital, and Professor, University of Chicago. Cloth. Pp. 999, with 406 illustrations. Springfield, Ill., 1937, Charles C. Thomas. $8.50.

The second edition of Grinker's *Neurology* is built up along the same logical plan as the first, with emphasis well laid on the embryology, anatomy, and physiology of the various units of the nervous system as an introduction to a study of their pathology and abnormal function in disease. The anatomic figures, cross-section drawings, and photomicrographs leave little to be desired. Succeeding chapters take up the spinal fluid, the functions of the motor unit, reflex activity, and sensation on which is based the technique of neurologic examination. In a similar way careful descriptions of cerebellar function, muscle tone, the extrapyramidal motor system, and the cerebral cortex precede the more purely clinical portions of the book.

From a neurosurgical viewpoint, the chapter on intracranial tumors, written with the assistance of Dr. Percival Bailey and Dr. Paul C. Bucy and based on the extensive material to be found in the University of Chicago Clinics, is thoroughly satisfactory. The descriptions of peripheral nerve and spinal cord tumors are also good.

In the account of trigeminal neuralgia the author states that "Frazier cuts the motor root for he fears that it may bear antidromic fibers to the mesencephalic nucleus and be responsible for those rare cases of persistent pain after the sensory roots have been cut." Inasmuch as Frazier was primarily responsible for the modern operation of selective section to spare the motor root, the reason for this statement is difficult to explain. There are numerous minor inaccuracies in the chapter on the vegetative nervous system, such as statements that the pharmacologic rôle of ergotamine is to stimulate the sympathetic fibers and that the vagus constricts the gut and produces peristalsis, but relaxes the sphincters at the pylorus and cecum. The work of Cannon, Alvarez, and many others has shown that the vagus carries both stimulator and inhibitor fibers, so that no such clear-cut response will ordinarily ensue. In the section on Raynaud's disease the selection of patients suitable for operation is not adequately described, and the discussion of the best surgical method to secure relief of vasospasm seems unnecessarily vague. Furthermore, in the light of recent experimental work, the diagrams of heart and bladder innervation (Figs. 96 and 98) are inaccurate. In Fig. 96 the direct cardiac nerves, which are so important in surgical intervention of angina pectoris, are omitted, while from Fig. 98 the reader would derive the view that there is a balanced sympathetic inhibitor-parasympathetic motor tonus of the detrusor vesicae. The work of Denny-Brown, of Evans, and of Munro has clearly shown that there is no such balanced control of bladder activity. To the neurosurgeon who has treated traumatic epilepsy on the lines laid down by Penfield, the author may seem over-conservative when he states that: "I have not seen this procedure [extirpation of a cortical scar] act to stop convulsions if all sedative medication such as luminal is withdrawn."

In comparison with other textbooks of neurology, which lack the excellent introductory chapters of this volume, it must be admitted that differential diagnosis and treatment of certain diseases do not stand out with the clarity of the preliminary anatomic and physiologic sections. An additional criticism is that numerous important clinical references have been omitted. For instance, in the chapter on muscular atrophies no mention is made of the differential test for myasthenia gravis with prostigmin which was proposed in 1935 by Viets and Schwab. Mention of this omission must be made because the test is of the greatest value in ruling out such conditions as the chronic progressive ophthalmoplegia of von Graefe and bulbar poliomyelitis, with which myasthenia gravis is often confused.

Taken as a whole the text is most readable and gives a logical and clear-cut understanding of the function of the nervous system in both health and disease. The index is complete and satisfactory. In the reviewer's opinion the sound anatomic and physiologic background of the work more than outweighs the clinical shortcomings which have been pointed out above. The book is not for the specialist who requires the complete picture and the most up-to-date information on clinical neurology, but it can be recommended enthusiastically for the medical student and the general practitioner who require a single volume text on the fundamentals of neurologic disease.